VOLUME 69 • JANUARY 1959 • NUMBER 1

A.M.A. ARCHIVES OF OTOLARYNGOLOGY

Chelation of Stapes Footplate
Joseph Sataloff

The Treatment of Ménière's Disease with Ultrasonic Waves
Franz Altmann and Jules G. Waltner

Sudden Deafness Due to Virus Infection
John R. Lindsay

Cancer of the Retromolar Area
Jorge Fairbanks Barbosa

Congenital Micrognathia
Chester M. Weseman

Safe Anesthesia for Peroral Endoscopy
William P. Kleitsch

Neurinoma (Schwannoma) of the Facial Nerve
Jack W. Pou

True Cholesteatoma
James L. Sheehy

Voice Therapy
F. Bertram Briess

Tympanic Skin Grafting and Reconstruction of the Middle Ear Sound-Conduction Mechanism
Frederick R. Guilford, William K. Wright, and W. Leonard Draper

Modifications of Cochlear Responses by Oxygen Deprivation
César Fernández and René Alzate

Congenital Stenosis of the Nasal Passages
Byron G. McKibben

Evaluation of the Sierra-Sheldon Tracheotome
H. Haskell Ziperman and Spencer P. Thornton

Otitis Media and Complications
B. R. Dysart

Abstracts from Current Literature

Books

Directory of Otolaryngological Societies

AMERICAN MEDICAL ASSOCIATION PUBLICATION

storz
Surgical Instruments

MICROSCOPE

for Mobilization of Stapes, Tympanoplasty and Endotympanic Surgery

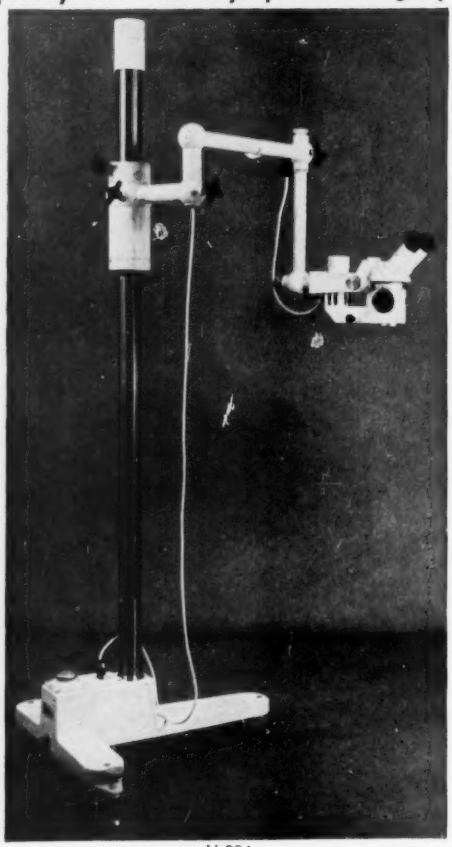
Binocular magnification and depth of focus affords stereoscopic observation resulting in a perception of depth comparable to natural vision even at 40 X.

Free working distance, or focal length, of 8 inches in all magnifications permits unobstructed surgery.

Brilliant illumination within the cavity by the light being directed to the field through the microscope objective. Any portion of the cavity which can be viewed will be illuminated.

Rapid magnification selector is a dial knob which rotates the lens drum through five changes of magnification. A change of power does not change the focus distance and can be made without interruption of observation. Microscope can be supplied with 12.5 X oculars for a range of 4, 6, 10, 16, and 25 X magnification and/or with 20 X oculars for a range of 6, 10, 16, 25, and 40 X. With either the working distance is 8".

Transformers for photo flash and fittings for mounting of photo lens system are included. Other photographic equipment is not supplied as standard equipment, but is listed below and can be ordered separately. All models supplied with two extra bulbs and for use with 110-115 volt cycle current.



N-994

N-992 **Microscope, Endaural Surgery, LEMPERT:** as described with 12.5 X oculars only and inclined binocular tubes as illustrated. **$1912.50**

N-993 Same as N-992, but with straight binocular tubes as used by Prof. Wullstein. Not illustrated. **$1906.50**

N-994 Same as N-992, but with both 12.5 X and 20 X oculars and inclined binocular tubes as illustrated. **$1990.50**

N-995 Same as N-994 but, with straight binocular tubes. Not illustrated. **$1984.50**

N-996 **Photographic Lens System and Electronic Flash Illumination** for all models. This equipment is not supplied with basic microscope, but can be ordered for delivery with microscope or separately at a later date. No tools required for installation. ..... **$ 356.00**

N-997 **Camera Body, CONTAX II-A:** consists of complete camera but without the lens which is part of the N-996 lens system. ..... **$ 179.00**
Excise Tax **20.00**

N-998 **Drape, Sterilizable:** for all models. ..... **$ 6.00**

4570 Audubon **STORZ INSTRUMENT CO.** **St. Louis 10, Mo.**

New York Showroom: 809 Lexington Ave.

TABLE OF CONTENTS

VOLUME 69 JANUARY 1959 NUMBER 1

ORIGINAL ARTICLES

PAGE

Chelation of Stapes Footplate
Joseph Sataloff, M.D., Philadelphia 1

The Treatment of Ménière's Disease with Ultrasonic Waves
Franz Altmann, M.D., and Jules G. Waltner, M.D., New York 7

Sudden Deafness Due to Virus Infection
John R. Lindsay, M.D., Chicago 13

Cancer of the Retromolar Area
Jorge Fairbanks Barbosa, M.D., Sao Paulo, Brazil 19

Congenital Micrognathia
Chester M. Weseman, M.D., Berkeley, Calif 31

Safe Anesthesia for Peroral Endoscopy
William P. Kleitsch, M.D., Omaha 45

Neurinoma (Schwannoma) of the Facial Nerve
Jack W. Pou, M.D., Shreveport, La. 48

True Cholesteatoma
James L. Sheehy, M.D., Los Angeles 57

Voice Therapy
F. Bertram Briess, Syracuse, N. Y. 61

Tympanic Skin Grafting and Reconstruction of the Middle Ear Sound-Conduction Mechanism
Frederick R. Guilford, M.D.; William K. Wright, M.D., and W. Leonard Draper, M.D., Houston, Texas 70

AUDIOLOGY SECTION

Modifications of Cochlear Responses by Oxygen Deprivation
César Fernández, M.D., and René Alzate, M.D., Chicago 82

Congenital Stenosis of the Nasal Passages
Col. Byron G. McKibben (MC), U. S. Army 95

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

Evaluation of the Sierra-Sheldon Tracheotome
Lieut. Col. H. Haskell Ziperman (MC), U. S. Army, and Capt. Spencer P. Thornton (MC), Army U. S. 98

PROGRESS IN OTOLARYNGOLOGY

Otitis Media and Complications
B. R. Dysart, M.D., Pasadena, Calif. 103

REGULAR DEPARTMENTS

Abstracts from Current Literature 119

Books:
Books Received 127
Book Reviews 127

Directory of Otolaryngological Societies 129

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 69 JANUARY 1959 NUMBER 1

COPYRIGHT, 1959, BY THE AMERICAN MEDICAL ASSOCIATION

EDITORIAL BOARD

GEORGE M. COATES, Chief Editor
1721 Pine Street, Philadelphia 3

RALPH A. FENTON, Portland, Ore.
ROBERT L. GOODALE, Boston
VICTOR GOODHILL, Los Angeles
CHEVALIER JACKSON, Philadelphia
JOHN D. KERNAN, New York
CARL H. McCASKEY, Indianapolis
JOSEPH SATALOFF, Philadelphia
ERNEST M. SEYDELL, Wichita, Kan.
GEORGE E. SHAMBAUGH JR., Chicago

J. F. HAMMOND, Editor, A. M. A. Scientific Publications
GILBERT S. COOPER, Managing Editor, Specialty Journals

SUBSCRIPTION RATES

Price per annum in advance, including postage: Domestic, $14.00. Canadian, $14.50. Foreign, $15.50. Price to students, interns, and residents, $8.00 in U. S. & possessions.

Single copies of this and previous calendar year, $1.50.

Back issues older than two years are available through Walter J. Johnson, Inc., 111 Fifth Avenue, New York 3. Future reprints of back issues will be available through Johnson Reprint Corporation, 111 Fifth Avenue, New York 3.

Checks, money orders, and drafts should be made payable to the American Medical Association, 535 North Dearborn Street, Chicago 10.

AMERICAN MEDICAL ASSOCIATION Publication

Published monthly by the AMERICAN MEDICAL ASSOCIATION. Editorial and Circulation Offices: 535 North Dearborn Street, Chicago 10, Illinois. Publication Office: Thompson Lane, Box 539, Nashville 1, Tennessee. Second-class mail privileges authorized at Nashville, Tenn., Aug. 6, 1956.

CHANGE OF ADDRESS: When there is a change of address, the Circulation Office of the American Medical Association should be notified at least six weeks before the change is made. The address label clipped from the subscriber's latest copy of the publication and a statement of old and new address should be included. If there is a postal zone number, it too should be included in the new address. The instructions should state whether the change is permanent or temporary.

cerumenex T.M.

(CONTAINS CERAPON* 10.0% IN PROPYLENE GLYCOL WITH CHLORBUTANOL 0.5%) DROPS

clinically proven†

93.5 PER CENT EXCELLENT RESULTS OBTAINED IN SEPARATE CONTROLLED SERIES COMPRISING 785 PATIENTS (ALL AGES) WITH EXCESS OR IMPACTED CERUMEN

CERUMENEX emulsifies and loosens earwax, facilitating its painless removal—without irritation or instrumentation—by simple syringing, flushing, or washing of ear with lukewarm water.

- effective, convenient, and painless; greatly expedites office procedures and saves time; simplifies routine aural examinations
- non-irritating and non-allergenic; well tolerated by patients in all ages
- helps relieve symptoms of itching, pain, autophony, impaired hearing, a sense of fullness, tinnitus and vertigo, if due to excessive cerumen
- hygroscopic—helps absorb aqueous transudates
- a specially-prepared water soluble agent specifically for facilitating removal of cerumen

DIRECTIONS FOR USE: Fill ear canal with CERUMENEX. Insert cotton plug and allow to remain overnight. (In some patients 15 to 20 minutes may be sufficient.) Then, gently flush or wash ear with soft rubber syringe using lukewarm water (avoid excessive pressure). If a second application is necessary in unusually hard impactions, repeat dosage and allow again to remain in situ overnight; then, repeat cleansing procedure. SUPPLY: 15 cc. bottle, packaged with cellophane wrapped blunt-end ear dropper.

*BRAND OF TRIETHANOLAMINE POLYPEPTIDE OLEATE-CONDENSATE U.S. AND FOREIGN PATENTS PENDING

†COMPLETE INFORMATION AVAILABLE UPON REQUEST TO THE MEDICAL DIRECTOR

PF *The Purdue Frederick Company* DEDICATED TO PHYSICIAN AND PATIENT SINCE 1892
NEW YORK 14, N.Y. | TORONTO 1, ONTARIO

© Copyright 1958, The Purdue Frederick Company

Instructions to Contributors

Articles, book reviews, and other materials for publication should be addressed to the Chief Editor. Articles are accepted for publication on condition that they are contributed solely to this journal.

An original typescript of an article, with one carbon copy, should be provided; it must be double or triple spaced on one side of a standard size page, with at least a 1-inch margin at each edge. Another carbon copy should be retained by the author.

The main title of an article may not contain more than eighty characters and spaces; a subtitle may be of any length.

The author's name should be accompanied by the highest earned academic or medical degree which he holds. If academic connections are given for one author of an article, such connections must be given for all other authors of the article who have such connections.

If it is necessary to publish a recognizable photograph of a person, the author should notify the publisher that permission to publish has been obtained from the subject himself if an adult, or from the parents or guardian if a child. An illustration that has been published in another publication should be accompanied by a statement that permission for reproduction has been obtained from the author and the original publisher.

Oversized original illustrations should be photographed and a print on glossy paper submitted. Prints of a bluish tinge should be avoided. Large photomicrograph prints will be reduced in scale unless portions to be cropped are indicated by the author. The author should submit duplicate prints of roentgenograms and photomicrographs with the essential parts that are to be emphasized circled, as a guide to the photoengraver.

Charts and drawings should be in black ink on hard, white paper. Lettering should be large enough, uniform, and sharp enough to permit necessary reduction. Glossy prints of x-rays are requested. Paper clips should not be used on prints, since their mark shows in reproduction, as does writing on the back of prints with hard lead pencil or stiff pen. Labels should be prepared and pasted to the back of each illustration showing its number, the author's name, and an abbreviated title of the article, and plainly indicating the top. Charts and illustrations must have descriptive legends, grouped on a separate sheet. Tables must have captions. ILLUSTRATIONS SHOULD BE UNMOUNTED.

References to the literature should be limited to those used by the author in preparation of the article. They should be typed on a special page at the end of the manuscript. The citation should include, in the order given, name of author, title of article (with subtitle), name of periodical, with volume, page, month—day of month if weekly or biweekly—and year. References to books must contain, in the order given, name of author, title of book, city of publication, name of publisher, and year of publication.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street **Chicago 10**

for fever and pain / in infants and children

when your little patient

is burning with fever.....

Tempra®

Acetaminophen, Mead Johnson

syrup • drops

brings relief quickly...conveniently

- **well tolerated**
- **easy to give**
- **tastes good**

Mead Johnson

Symbol of service in medicine

TE603

an important problem in today's living...

The following articles from TODAY'S HEALTH are now available in pamphlet form.

ALCOHOLISM IS A DISEASE. A discussion by the Chairman of the A.M.A. Committee on Alcoholism. by Marvin A. Block, M.D., 8 pages, 15 cents.

I AM THE WIDOW OF AN ALCOHOLIC. Three articles combined. by Virginia Conroy, 16 pages, 20 cents.

HOW EXPERTS MEASURE DRUNKENNESS. A partial transcript of an actual courtroom case. by H. A. Heise, 8 pages, 15 cents.

BARBITURATES, BOOZE AND OBITUARIES. A discussion of the dangers of mixing alcohol and barbiturates. by Donald A. Dukelow, 4 pages, 10 cents.

TWELVE STEPS FOR ALCOHOLICS. A frank discussion of the meaning of an alcoholic behavior. by Richard Lake, 6 pages, 10 cents.

These articles are available in one pamphlet for 50c

ALCOHOLICS ANONYMOUS. Written from the standpoint of a member, the basic treatment procedures are described and the psychological problems confronting the alcoholic are discussed.

ALCOHOL AND CIRRHOSIS OF THE LIVER. Relationship between alcohol, diet and cirrhosis. Increasing stress on nutritional differences. by Russell S. Boles.

HOW TO HELP A PROBLEM DRINKER. Understanding the alcoholic's capabilities, the necessity of help, causes of his condition. by Edward A. Strecker and Francis T. Chambers, Jr.

THE TREATMENT OF ALCOHOLISM. Tracing the steps from convincing the alcoholic that he is sick through treatment and cure. by Lewis Inman Sharp.

CONDITIONED REFLEX TREATMENT OF CHRONIC ALCOHOLISM. Its place among methods of treatment today, its development and correlation with personality factors. by Walter L. Voegtlin.

INSTITUTIONAL FACILITIES FOR THE TREATMENT OF ALCOHOLISM. Comparative differences, in drinking, with the last century, new establishments and methods of treatment, lack of trained personnel. by E. H. L. Corwin.

ADDRESS REQUESTS TO

ORDER DEPARTMENT

AMERICAN MEDICAL ASSOCIATION

535 N. DEARBORN ST., CHICAGO 10, ILLINOIS

THE UNIVERSITY OF MICHIGAN MEDICAL SCHOOL

Postgraduate Conference in Otolaryngology

The annual conference in Otolaryngology will be held April 16, 17 and 18, 1959, at the University Hospital, Ann Arbor, Michigan.

The conference will be conducted by Dr. James H. Maxwell and Staff of the Department of Otolaryngology, together with eminent guest lecturers in the field of Otolaryngology.

Application and fee of $50 may be addressed to the Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

731/S

RHINOPLASTIC INSTRUMENTS
Designed By **DR. B. B. RISH**

Cat. No. 731/S RISH OSTEOTOME (Original Model) Stainless Steel, Overall Length 7" (18 cm.) $9.50 ea.

Designed for Intranasal Osteotomy.

Features. One instrument for both sides —Short, blunt, nonbreakable guard-guide, manually felt through skin follows exact osteotomy course. A Collar (ledge) over dorsal aspect which prevents slipping medially or laterally. Uniform thickness of 1 mm for 1½" (38 mm) from cutting edge to prevent separation of nasal fragments with premature low Fracture. Ref. Archives of Otolaryngology, Feb. 1953.

Cat. No. 717/S RISH CARTILAGE KNIFE, STAINLESS, $9.75 ea. Designed for "shaving" dorsal cartilage in Rhinoplasty.—Regardless of pressure exerted, it is impossible to remove more than a paper-thin portion of Cartilage.—

Ref. Archives of Otolaryngology, Oct. 1954.

Write for current Catalog No. 18.

B. J. FLORSHEIM
2067 Broadway, at 72nd. St.
New York 23, N. Y.

717/S

For a three-dimensional concept . . .

Wolff et al.:

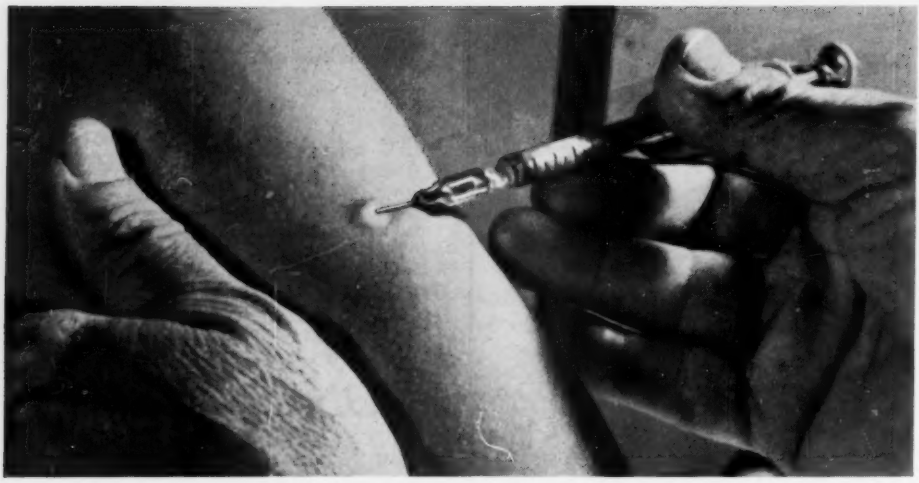
MICROSCOPIC ANATOMY OF THE TEMPORAL BONE

"It is common knowledge that few laboratories can provide microscopic sections of the temporal bone for study, and the literature on the subject is not an adequate source of teaching material. So rapid and fascinating have been the advances in this field in recent years that a desire has been aroused among otologists to study this organ in greater detail. An intimate knowledge of the topographic microscopic anatomy is absolutely essential for the surgeon to obtain a three dimensional concept of the temporal bone in order to perfect the surgical techniques for conservation of hearing. Under such circumstances, this profoundly illustrated monograph on the subject written on modern lines embodying the latest advances, fulfils a badly needed current want in the literature on the subject."—*J. Indian M. Prof.*

CONTENTS—Series in Horizontal Plane (*63 photomicrographs*). Vertical Series Cut at Right Angles to Long Axis of Petrosa (*92 photomicrographs*). Vertical Series Cut in the Plane of Long Axis of Petrosa (*37 photomicrographs*). Addendum (*8 photomicrographs of monkey organ of Corti*).

By DOROTHY WOLFF, Ph.D., *Research Pathologist . . .*; RICHARD J. BELLUCCI, M.D., *Surgeon-in-Charge of Speech and Hearing Clinic . . .*; *and* ANDREW A. EGGSTON, M.D., *Pathologist and Director of Laboratories . . .*; *all from Manhattan Eye, Ear and Throat Hospital* (1957). 414 pp., 198 photomicrographs. **$12.50**

THE WILLIAMS & WILKINS COMPANY
Mount Royal & Guilford Aves., Baltimore 2, Md.



when allergy tests are positive for

· food · dust · detergents · pollen · drugs

'Teldrin' provides "round-the-clock" relief from these "round-the-year" allergies.

Just one 'Teldrin' *Spansule* capsule q12h provides rapid, effective and prolonged relief from allergic symptoms. Side effects from 'Teldrin' therapy are minimal.

Available in 2 dosage strengths: 12 mg. for adults and older children; 8 mg. for younger children.

Teldrin* chlorprophenpyridamine maleate

Spansule* sustained release capsules, S.K.F.

SMITH KLINE & FRENCH LABORATORIES

*T.M. Reg. U.S. Pat. Off.

A.M.A. ARCHIVES OF
OTOLARYNGOLOGY

Chelation of Stapes Footplate

JOSEPH SATALOFF, M.D., Philadelphia

A chelating agent is a chemical compound capable of forming a very stable complex with a multivalent positive ion, such as calcium. In this process of chelation a specific type of coordinate bonding occurs in some of the atoms forming the ring, and the calcium becomes such an integral part of the complex that it loses its ionic properties. It cannot be precipitated by generally used precipitating agents.[1]

Edathamil (ethylenediaminetetraacetic acid [EDTA]) and its salts have been the most investigated and commonly used chelating agents. Figure 1A shows the acid to be tetrabasic with four replaceable hydrogen atoms, so that it is theoretically possible to form four different sodium salts, all of which are chelating agents. Figure 1B shows the disodium salt of edathamil and 1C shows the calcium-disodium salt, which is the end-product of the chelation of the calcium ion. In this study, the disodium salt of edathamil was usually used.

The chelating properties of this and of other agents depend to a great degree upon

Submitted for publication June 25, 1958.

From the Department of Otolaryngology, Jefferson Medical College.

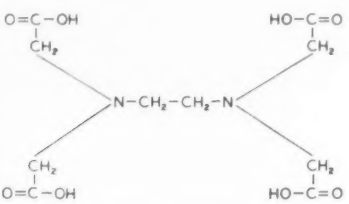


Fig. 1A.—Edathamil (ethylenediaminetetraacetic acid).

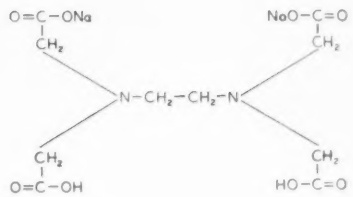


Fig. 1B.—Edathamil disodium (disodium ethylenediaminetetraacetic acid).

the pH of the solution, which determines the amount of ionization, and upon the stability constant, which expresses the tenacity of the particular bond in the complex.[2] Chelating agents have been used to treat patients with lead poisoning,[1] to remove corneal calcium deposits,[3] and to dissolve urinary calculi (experimentally).[4]

Edathamil produces no apparent adverse effects upon human beings when slowly administered intravenously daily for a week or intermittently for several weeks.[1] A pilot experiment conducted on postmortem stapedes and the middle ears of living patients indicated that a pH of 7.3 or 7.4 was satisfactory for the present investigation.

Fifty stapedes were removed from fresh autopsy specimens and were placed in normal saline. None of the stapedes demonstrated otosclerotic fixation grossly and all were from persons over 40 years of age. Twenty stapedes were used as controls and the others as an experimental group. The most commonly used procedure was to place a saturated wick of absorbent cotton directly upon the superior surface of the footplate but not in contact with the crura.

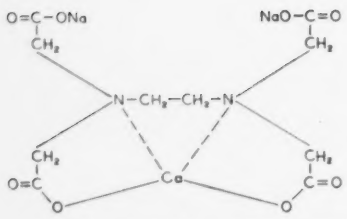


Fig. 1C.—Edathamil calcium-disodium (calcium-disodium ethylenediaminetetraacetic acid).

In the experimental group, the wick was then continually saturated with edathamil disodium by means of an eye dropper; in the controls the wick was saturated with normal saline. Other techniques consisted of immersing the entire stapes (as well as some mallei and incudes) in a solution of edathamil disodium, wrapping saturated wicks around the crura, and placing the footplates in puddles of the agent. In many instances, the procedure was repeated after the mucosa had been removed from the stapes. Some of the stapedes along with their controls were kept in a watch glass at room temperature, while others were maintained at normal body temperature in an incubator. Figure 2 shows the wick in place between the crura upon the footplate.

Results

In none of the 20 control stapedes was there any observable change in any pertinent characteristic after treatment with normal saline. In the experimental group of stapedes there was a change in every one. Invariably the portion bathed with the chelating agent became soft, pliable, elastic, decalcified, and could readily be penetrated or excavated by gentle pressure with a sharp pick. In stapedes that were immersed these effects were observed on the entire bone; where only the footplate was treated, only the footplate was affected. In the bones that were immersed completely, there was a tendency for certain parts of the footplate to become decalcified before the crura.

Figure 3 shows a normal untreated stapes which did not differ noticeably from those treated with normal saline.

Fig. 2.—Normal stapes with wick on footplate.

Fig. 3.—Normal stapes.

Fig. 4.—Stapes immersed in edathamil disodium for six days.

Fig. 5.—Stapes immersed in edathamil disodium for nine days.

Figure 4 shows a stapes that has been treated by complete immersion in 10% edathamil disodium for six days. While the magnitude of change and the alteration in texture were not as apparent in a photograph as in the bone itself, it is nevertheless clear that this stapes is thinner than the untreated one, particularly in the footplate. Before three hours of immersion had elapsed the entire stapes became pliable and elastic, similar to a thin bone after being immersed in vinegar. Except for appearing thinner and decalcified the stapes did not show any change in shape after six days of chelation. Even after nine days of immersion (Fig. 5) the stapes maintained its normal shape when handled with care during inspection.

Gentle pressure on any portion of the stapes caused depression and distortion, but the shape returned to normal when the pressure was removed. With greater pressure, a permanent disfigurement resulted (Fig. 6).

Changes in the footplate are of particular interest because of the role of the footplate in clinical otosclerosis. The thinning appeared much earlier and was more pronounced at the outer edges of the footplate midway between the crura than directly in the center of the footplate. In the early stage of decalcification it was possible by firm pressure to distort the edges of the footplate into peculiar configurations which persisted when the stapes dried after removal from the chelating agent (Fig. 7, distorted stapes footplate which had been exposed to edathamil [Versenate] wick for 24 hours).

After the stapes was removed from the chelating agent, it remained flexible for several days when kept in a tightly closed container; when exposed to the air it became stiff and dry. It was, however, considerably thinner and lighter and obviously decalcified.

Figure 8 shows a stapes upon which a wick of 10% edathamil disodium was placed on the footplate so that it also en-

Fig. 6.—Stapes permanently distorted by pressure after stapes has been decalcified.

Fig. 7.—Stapes with distorted footplate after decalcification with a wick saturated in edathamil solution.

Fig. 8.—Stapes with humped footplate following decalcification with edathamil disodium wick.

Fig. 9.—Stapes footplate exposed to wick of edathamil for 24 hours.

compassed the posterior aspect of the base of the anterior crus. After three hours there was definite softening at the outer edges of the exposed area on the footplate. After five hours' exposure the entire footplate became soft and flexible, and any change of shape by firm pressure remained permanent. This figure shows the distortion accomplished by gentle pressure upwards in the middle of the footplate. Note that this footplate is still intact and not cracked or broken. The edges were softer and more pliable than the center. The anterior and posterior parts of the footplate were much firmer and less affected, so that the middle gave more easily. There was some mild softening at the area exposed behind the anterior crus. The stapes shown is bone-hard and is dry. Neither of the crura was affected.

Figure 9 shows a footplate exposed for 24 hours in 5% sodium edathamil (Versenate) and maintained at 37.5 C in an incubator. The footplate in the area exposed has become greatly thinned and pliable, particularly at the edges. The rest of the stapes has not been affected.

Fig. 10.—Stapes footplate exposed to wick of edathamil for 48 hours.

Figure 10 shows a footplate exposed in a manner similar to that in Figure 9 except that the exposure time was 48 hours. Here it is quite evident the edges of the exposed footplate have become so thin and decalcified they are hardly visible except as strands of connecting tissue. It is interesting to note here that the stapes footplates treated by saturated wicks showed more thinning and destruction than those that were completely immersed in the chelating agent. This may be due to the fact, however, that the investigator had a tendency to feel the former footplates much more frequently than the latter to test the firmness and flexibility.

Figure 11A shows a normal malleus, and Figure 11B shows a malleus immersed in a 3% solution of sodium edathamil for 112 hours. After 36 hours the malleus was completely flexible and decalcified. The only bony area palpable was a small spot at the head. No parts of the malleus eroded, for its shape was maintained by a thin bony framework including firm connective tissue.

All of the stapedes exposed to chelation showed results similar to those demonstrated in these figures. The changes in texture, size, pliability, et cetera, are of course much more apparent in the gross specimens than in the photographs.

Comment and Conclusions

Quite obviously, those ossicles treated with a chelating agent showed a substantial weight loss and were markedly thinned and decalcified. Significant changes were observed only in the areas exposed to the agent; the rest of the bone seemed unaffected. There was little difference between the stapedes with mucosa and those from which the mucosa was removed, but this was attributable to the fact that the mucosa was usually torn and shredded.

Fig. 11A.—Normal malleus.

Fig. 11B.—Malleus immersed in edathamil for 112 hours.

Within limits, the degree of decalcification was a direct function of the length of exposure. It is noteworthy that even when a stapes was immersed for many days it retained its normal shape except for the pronounced decalcification at the edges of the footplate.

Generally, the footplate was more readily decalcified than other parts of the stapes, and the edges of the footplate usually were the first to soften. The implications of this with respect to clinical otosclerosis are obvious. The thin anterior crus took slightly longer and the posterior crus considerably longer to become decalcified. The head of the stapes remained hard long after other parts had been softened.

In addition to the present experiment on postmortem stapedes, in vivo investigations on animals and on humans have been in progress for the past two years. While it is still too early to make a definitive report, the use of edathamil disodium in the treatment of patients with clinical otosclerosis seems worthy of further investigation.

The following during the past two years have aided this investigation: Dr. John Zapp, Dr. G. M. Coates, Dr. Fred Harbert, Mr. Henry Hoff, Dr. H. Menduke, The Iso Sol Company, The Dow Chemical Company, Dr. R. P. Gwinn of Abbott Laboratories, and Mr. Phillip VanItalie.

1721 Pine St. (3).

REFERENCES

1. Spencer, H.; Vankinscott, V.; Lewin, I., and Laszlo, D.: Removal of Calcium in Man by Ethylenediamine Tetraacetic Acid: A Metabolic Study, J. Clin. Invest. 31:1023-1027 (Dec.) 1952.

2. Chenoweth, M. B.: Chelation as a Mechanism of Pharmacological Action, Pharmacol. Rev. 8:57-87 (March) 1956.

3. Grant, W. M.: New Treatment for Calcific Corneal Opacities, A. M. A. Arch. Ophth. 48:681-685 (Dec.) 1952.

4. Gehres, R. F., and Raymond, S.: A New Chemical Approach to the Dissolution of Urinary Calculi, J. Urol. 65:474-83 (March) 1951.

5. Clarke, N. E.; Clarke, C. N., and Mosher, R. E.: The "in Vivo" Dissolution of Metastatic Calcium: An Approach to Atherosclerosis, Am. J. M. Sc. 229:142-149 (Feb.) 1955.

The Treatment of Ménière's Disease with Ultrasonic Waves

A Preliminary Report

FRANZ ALTMANN, M.D., and JULES G. WALTNER, M.D., New York

In spite of considerable progress during the last 20 years, the results of the medical treatments of Ménière's disease are still far from being satisfactory. Surgical destruction of the diseased labyrinth is, furthermore, feasible only in the limited number of cases in which in all probability only one labyrinth is affected and in which the hearing on the affected side has permanently fallen below the serviceable level. The numerous cases which do not respond to medical treatment and which, on the other hand, are not suitable for surgery present a very difficult therapeutic problem.

A new treatment aiming at selective destruction of the diseased vestibular portion of the labyrinth with ultrasonic waves with preservation of hearing would be ideally suited for these patients.

Although attempts at treatment of the ear with ultrasonic waves date back as far as 30 years, the apparatus used at that time by Mülwert and Voss produced waves of such low intensity that they could not reach the inner ear at all. Later, machines capable of producing ultrasonic waves of higher frequency and of much higher intensity were used (Wiethe, Wyt, and Vyslonzil and others). The application was first purely empirical, without any theoretical or experimental basis.

Submitted for publication April 2, 1958.

In part supported by a grant from the Hayden Fund for the Coakley Memorial Clinic.

From the Department of Otolaryngology, Presbyterian Hospital in the City of New York.

Read before the Section on Otolaryngology of the American College of Surgeons, New York, March 3, 1958.

Subsequently it was shown in animal experiments by Vyslonzil that ultrasound produces first a stimulation of the vestibular apparatus and that application of larger or repeated stimuli is followed by a complete loss of vestibular responses.

It was furthermore demonstrated that in man even ultrasounds of higher intensities when applied to the outer surface of the mastoid practically do not reach the inner ear, because they are almost completely absorbed by air and to a considerable extent by bone (Pfander, Frenzel and Pfander, Seidl and Krejci). Only in patients with sclerotic mastoids could a significant proportion of the ultrasound possibly reach the inner ear.

Animal experiments, together with histological studies, showed that if the ultrasound actually reaches the inner ear from the outside of the skull not only the vestibular but also the cochlear portion are damaged. Irradiation of the whole labyrinth with doses large enough to cause destructive changes in the endorgans did, under these circumstances, not present any appreciable advantages over the surgical destruction (Krejci, Portmann, Portmann and Barbe, Naumann).

Therefore, Krejci in other experiments, after operative exposure of the inner ear, tried to irradiate selectively the vestibular portion of the inner ear only. Using a narrow beam of ultrasonic waves, he succeeded in eliminating vestibular function with preservation of cochlear function. Encouraged by these experimental results, Krejci then tried to destroy the vestibular

portion of the labyrinth in a patient suffering from Ménière's disease. An antrotomy was performed, the semicircular canals skeletonized and irradiated with a beam with a diameter of 4 mm. by direct application of a specially constructed soundhead to the canals. Seven days after the irradiation a facial paralysis was noted. Eleven months after the operation the attacks of vertigo and the tinnitus had completely, and the facial paralysis almost completely, disappeared. The hearing on the treated side had decreased by 10 db. and the caloric vestibular responses were completely absent.

Arslan perfected Krejci's method by constructing, together with Federici, a thin and narrow sound-wave emitter which produces a narrow beam of sound waves with a frequency of 800,000 to 1,000,000 cps and of an intensity sufficient to produce a relatively rapid destruction of the vestibular portion of the inner ear on application to the bony wall of the lateral semicircular canal. Practically no sound waves seem to reach the medial wall of the petrous bone, and the posterior cranial fossa. No ultrasound waves can escape from the side walls of the emitter, and thus facial paralysis or further hearing loss can be prevented.

Arslan proceeds in the following way: With the area under local anesthesia, an opening is made into the antrum by a short retroauricular incision and enough cells are removed to get a good exposure of the lateral semicircular canal without disturbing the incus. Very careful hemostasis is essential, since even a small quantity of blood around the tip of the ultrasound emitter would increase the field of irradiation. The tip of the emitter is applied to the convexity of the lateral semicircular canal (Fig. 1); the contact between the two should be as close as possible because intervening air would prevent the ultrasound waves from reaching the inner ear. The ultrasound should be applied in such a way that the facial nerve and the cochlea are not included in the field of irradiation (Fig. 2).

Nystagmus should be constantly observed by an assistant during application of the sound waves in order to determine the

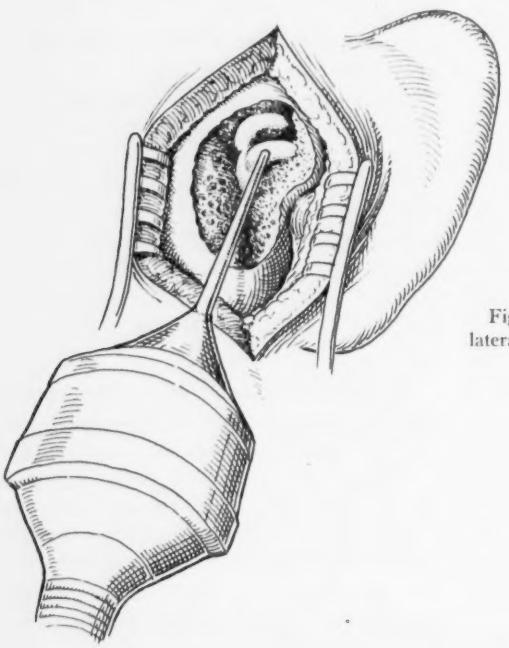


Fig. 1.—Tip of the sound head applied to the lateral semicircular canal.

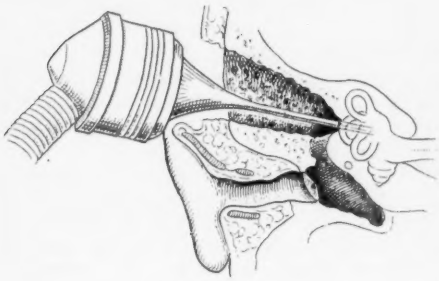


Figure 2

Fig. 2.—Beam of ultrasonic waves directed toward the vestibular portion of the labyrinth, avoiding the cochlea and the facial nerve.

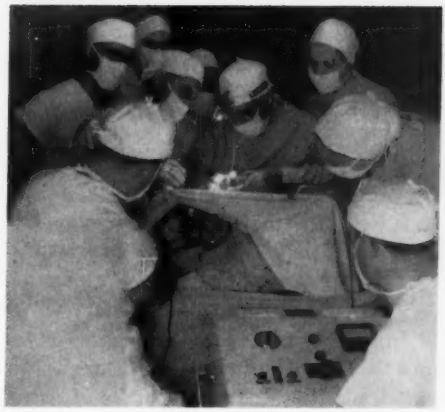


Figure 3

Fig. 3 (Case 4).—Operation. While the ultrasound is applied, an assistant observes the nystagmus and another assistant regulates the intensity of the ultrasound. Photograph by Dr. V. N. Epanchin.

various stages in the action on the vestibular system and the period required for their application. At the same time the assistant should watch out for any signs of facial nerve involvement (Fig. 3).

Nystagmus appears as early as within two to three minutes after the onset of the irradiation. Initially, it is "irritative" in type, i. e., its rapid component is directed toward the treated side. Some minutes later the direction of the nystagmus is reversed. It becomes "paralytic" in character, with the quick component toward the untreated side. The form of the nystagmus varies with the direction of the beam of ultrasound. For instance, when the waves are directed toward the vertical canals, the nystagmus will be either rotatory or vertical.

The doses of ultrasonic waves vary with the time of onset of the nystagmus and its subsequent course. Initially 7-8 watts/cm.^{2} are given; when the irritative nystagmus has disappeared, usually within 8-10 minutes, the dose is increased to 10-12 watts/cm.^{2}, until the paralytic nystagmus appears. When this form of nystagmus fails to appear, as in cases with markedly reduced vestibular responses, irradiation is continued until 25-30 minutes after the onset of the irradiation.

The *postoperative course* does, as a rule, not show the marked symptoms which are found after surgical destruction of the inner ear. The paralytic nystagmus usually disappears within 48 hours after the operation, but it usually takes 3-6 weeks before the dizziness goes away completely. Solitary attacks of vertigo may occur during the first weeks, but they are usually less severe than the preoperative attacks. After three to four months the unsteadiness after sudden movements of the head disappears.

The majority of Arslan's patients showed a marked improvement in hearing in the lower and middle tonal range. The tinnitus improved in the majority of cases; in many it was completely eliminated.

Experiments in dogs showed that the type of radiation used by Arslan selectively destroys the neurosensory epithelium of the cristae ampullares.

Arslan doubts that the initial "irritative" nystagmus is solely a hot-caloric nystagmus from the heat created by the ultrasound and feels that a direct mechanical vibratory effect of the ultrasound on the sensory neuroepithelium should also be considered a definite possibility. The therapeutic effect of the irradiation could be due either to the destruction of the neuroepithelium of the crista or to a relief from the endolymphatic hydrops by reducing the hypersecretion or facilitating the resorption of the endolymph. It is in his opinion very well possible that

both factors play a role in bringing about the favorable results.

The improvement in tinnitus could also be due to the improved endolymphatic circulation in the vestibular as well as the cochlear portion of the labyrinth.

Arslan reports, without giving detailed statistics, that the attacks of vertigo have disappeared in 95% of his patients, some of whom had been followed for as long as two and one-half years after the operation.

In 4 of the 88 cases the vestibular portion of the labyrinth had not been completely destroyed and the attacks of vertigo had not completely ceased within one year after irradiation. Ultrasound was, therefore, applied a second time, and thereafter all the vertigo stopped.

In none of the cases of Ménière's disease had it been necessary for Arslan to apply ultrasound to both ears, in spite of the frequent involvement of both labyrinths in this disease.

The results reported by Arslan seemed very impressive, and after a visit to Arslan's department in Padua, Italy, by one of us (J. G. W.) it was decided to try this method of treatment in the E. N. T. Department of Presbyterian Hospital.

Although only six cases have so far been treated by us, we nevertheless feel that a presentation of our preliminary results might be of interest.

Report of Cases

Case 1.—A 57-year-old man. Attacks for four years, with perceptive hearing loss in left ear, averaging 60 db. for the conversational range.

June 3, 1957. Application of ultrasound to the left labyrinth.

Seven months after application. No more vertigo for five months; ringing has completely disappeared, the hearing is unchanged from the best it was between attacks.

Case 2.—A 67-year-old man. Attacks for about one year, with perceptive hearing loss in the right ear, averaging 80 db., and ringing.

June 3, 1957. Application of ultrasound to the right labyrinth.

Eight months after application. Attacks of vertigo much improved; unsteadiness between attacks persists; hearing unchanged; ringing persists. Caloric vestibular responses slightly hypoactive on the side operated on.

Case 3.—A 30-year-old woman. Attacks for one and one-half years, increasing in severity, with perceptive hearing loss in the right ear, averaging 40 db. and marked ringing.

Sept. 17, 1957. Application of ultrasound to the right labyrinth.

Five months after application. No more vertigo for more than three months, hearing loss now averaging 20 db.; ringing has almost completely disappeared. No vestibular responses on the side operated on.

Case 4.—A 72-year-old man. Attacks of vertigo for many years with bilateral perceptive hearing loss averaging 70 db. on the right and 80 db. on the left side, ringing in both ears, more marked on the left.

Sept. 26, 1957. Application of ultrasound to the left labyrinth (Fig. 3).

Five months after application. No more vertigo since the operation, hearing unchanged, ringing in left ear has almost completely disappeared. No vestibular responses on the side operated on.

Case 5.—A 51-year-old man. Attacks for six months with ringing in left ear; mixed hearing loss, left side, averaging 50 db. Patient states that simple mastoidectomy was done many years ago; drum shows dry posterosuperior marginal perforation.

Dec. 10, 1957. Exploratory modified radical mastoidectomy; no pathology found. Application of ultrasound to the left labyrinth.

Two and one-half months after application. Attacks persist, ringing continues, hearing unchanged. Prompt vestibular responses after insufflation of cold air into the external auditory meatus.

Case 6.—A 48-year-old woman. Attacks for six years with ringing in left ear and perceptive hearing loss, averaging 80 db.

Dec. 18, 1957. Application of ultrasound to the left labyrinth.

Two months after application. Attacks of diminishing intensity until one month ago, ringing markedly improved, hearing unchanged; vestibular responses absent.

Comment

A review of these six cases shows that the hearing remained practically unchanged in five of them and improved in one. We do, however, not believe that this improvement exceeds the fluctuations frequently noted in cochlear hydrops and we are wondering if this is not also the case in the

patients observed in Arslan. Of greater significance seems to us the fact that in none of our patients did the hearing get noticeably worse.

The beneficial effect of the radiation on the ringing was quite striking, and in this respect the irradiation seems superior to all other forms of conservative or surgical treatment.

Whether the vertiginous attacks have been completely eliminated or not can be decided only after a much longer period of observation. In two of the cases (2 and 5) the amount of ultrasound applied to the labyrinth had evidently been insufficient to destroy the vestibular portion. This explains the persistence of vertigo together with the persistence of the vestibular responses. In order to get a complete destruction of the vestibular end-organs, one should not be content with the appearance of the "paralytic" nystagmus in one particular position of the applicator; the direction of the beam of ultrasonic waves should then be changed, and one might in a different position again get "irritative" nystagmus. The application should not be terminated until one gets "paralytic" nystagmus in all positions.

The two just-mentioned cases will, therefore, require a second treatment with ultrasound. Increasing experience with this type of treatment will undoubtedly greatly reduce the number of such cases.

But even now we feel that the results of the treatment of Ménière's disease with ultrasound are so encouraging that it is not improbable that this method will not only replace the present surgical methods but that it will also be used in many cases in which at present medical treatment seems the only possibility.

Summary and Conclusions

The principle of the treatment of Ménière's disease with ultrasonic waves is discussed and the Arslan technique described.

The preliminary results in six cases treated with this method are quite promising. In four of them the attacks of vertigo were eliminated. The failure of the treatment in two cases is evidently due to an insufficient amount of ultrasound applied to the labyrinth. Increasing experience with the technique will undoubtedly greatly reduce the number of such cases.

10 E. 85th St.

BIBLIOGRAPHY

Arslan, M.: Treatment of Ménière's Syndrome by Direct Application of Ultra-Sound Waves to the Vestibular System, in Proceedings of the Fifth International Congress of Otolaryngology, Amsterdam, 1953. Assen, Netherlands, Van Gorcum, 1953.

Direkte Applikation des Ultraschalfs auf das knöcherne Labyrinth zur Therapie der Labyrinthose (Morbus Ménièri), HNO 4:166, 1954.

Neue Resultate der Applikation des Ultraschalles auf das Labyrinth: Ein Beitrag zur Therapie der Labyrinthose, Arch. Ohren-Nasen.- u. Kehlkopfh. 167:559, 1955.

Frenzel, H., and Pfander, F.: Über die Grundlagen der Ultraschalltherapie im Ohrgebiet, HNO f. 2:193, 1950.

Krejci, F.: Experimentelle Grundlagen einer extralabyrinthären chirurgischen Behandlungsmethode der Ménièreschen Erkrankung, Pract. oto-rhino-laryng. 14:18, 1952.

Die Bedeutung des Ultraschalles in der Ohrenheilkunde, Wien. klin. Wchnschr. 64:719, 1952.

Mülwert, H., and Voss, O.: Eine neue physikalische Behandlungsmethode chronischer Schwerhörigkeit und deren Ergebnisse, Arch. Ohren- Nasen- u. Kehlkopfh. 119:81, 1928.

Naumann, H.: Ultraschall und Ohr-Region, Arch. Ohren- Nasen- u. Kehlkopfh. 160:240, 1951.

Pfander, E.: Experimentelle und klinische Versuche über die Ausbreitung des Ultraschalles im Ohrgebiet und Schädel, Arch. Ohren-Nasen- u. Kehlkopfh. 160:32, 1951.

Portmann, G.; Portmann, M., and Barbe, L. J.: Étude expérimentale (fonctionelle et histologique) de l'action des ultra-sons sur l'audition, Acta otolaryng. (Suppr 100) p. 119, 1952.

Portmann, M., and Barbe, L. J.: Les Ultra-sons et les nerfs de l'oreille (étude morphologique), Arch. Anat. Hist. et Embryol. 34:361, 1952.

Seidl, F., and Krejci, F.: Untersuchungen über die Durchdringbarkeit des Schläfenbeins mit Ultraschall mittels der Schlierenmethode, Practica oto-rhino-laryn. 14:65, 1952.

Voss, O.: Ultraschallwellen im Dienste der Behandlung chronischer Schwerhörigkeit, Arch. Ohren- Nasen- u. Kehlkopfh. 135:258, 1933.

Vyslonzil, E.: Über die Beeinflussung des Vestibularapparates durch Ultraschall, Wien. klin. Wchnschr. 61:468, 1949.

Wiethe, C.; Wyt, L., and Vyslonzil, E.: The Therapeutic Use of Supersonic Waves, Acta otolaryng. (Supp. 78) p. 111, 1948.

Sudden Deafness Due to Virus Infection

JOHN R. LINDSAY, M.D., Chicago

The purpose of this presentation is to review one segment of a problem which often confronts the otologist, the etiologic diagnosis in cases of sudden deafness, with or without vertigo.

A sudden or rapid onset of deafness may occur at all ages, either with or without vertigo.

Certain circumstances associated with the onset may indicate the etiology in some cases. In others there may be evidence of the general nature of the disturbance, without any specific knowledge of the pathologic process. In many instances there is a lack of information both as to etiology and the pathologic process.

If we exclude from our discussion such well-established causes for sudden deafness as labyrinthitis due to bacterial infection, trauma, and tumor, certain general groups may be differentiated. The commonest of these are Ménière's disease (labyrinthine hydrops), vascular lesions, virus infections, and a group in which the etiology and pathology are quite obscure. There are also some less common types associated with disease of other organs or systems.

Virus Infection

The most classical examples of sudden deafness due to virus infection are those due to two acute contagious diseases of childhood, mumps and measles.

The occurrence of unilateral profound deafness as a complication of mumps is well known to otologists, but may easily escape detection in small children since it is usually painless and is not associated with other complications such as encephalitis. The discovery is usually made by accident when the child is older.

Older children or adults complain of the tinnitus and hearing loss, but the profound nature of the deafness is discovered only on testing the hearing while the good ear is masked adequately with a loud noise apparatus.

Although we have observed several cases of profound unilateral deafness due to mumps every year for many years, I have not seen a bilateral case. Dizziness has also been a rare occurrence. I have seen only one in which there was also severe vestibular involvement along with the deafness. In that instance there was constant vertigo at the onset, which gradually decreased, became a postural symptom only, but had persisted for about six weeks when last seen.

There is no case on record in which the pathology has been studied. The route of infection, however, probably is by way of the blood stream, a complication of a viremia, in view of the fact that there is no middle-ear involvement and the inner-ear complication is not associated with a meningoencephalitis.

Deafness due to the measles virus is less common than that from mumps, but has usually been bilateral. The degree of deafness varies, and the incidence in several institutions for the deaf has been estimated at from 3% to 6%. Deafness or impaired hearing due to a bacterial infection complicating the measles has been commoner. We were able to find histopathologic reports of the inner ear changes in measles deafness in only three cases previous to the report recently made by us.[1] Our case demonstrated that the measles virus had caused extensive damage to the structures within the cochlear duct in both ears (Fig. 1). The route of entry had apparently been by way

Submitted for publication April 14, 1958.

From The University of Chicago, Division of Otolaryngology.

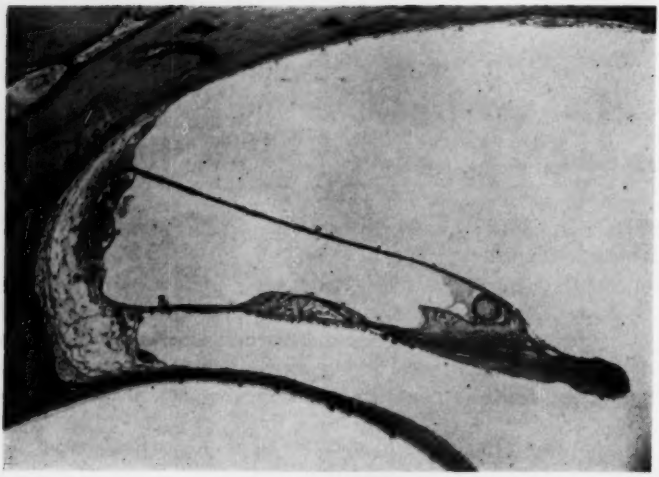


Fig. 1.—Section through the apical coil, left ear, showing the supporting structures of Corti's organ well preserved, but the hair cells degenerated and the tectorial membrane detached, rolled into a ball and lying in the angle between Reissner's membrane and the osseous spiral lamina. The stria vascularis also shows a degenerative change.

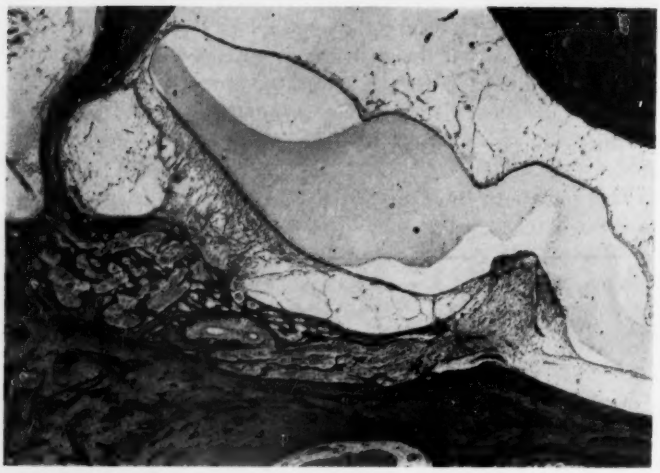


Fig. 2.—Section through upper part of the utricular macula and the crista ampullaris of the horizontal semicircular canal. The endolymph shows increased affinity for eosin. The otolithic membrane and the cupula are absent. The sensory epithelium has degenerated. On both sides of the crista can be seen a proliferative mass which consists mainly of young fibrous connective tissue, small lymphocytes with the degenerated sensory epithelium beneath. The infiltration extends throughout the crista as well.

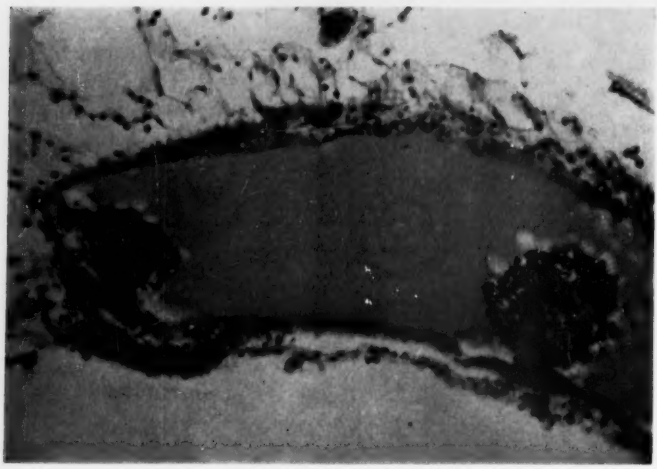


Fig. 3.—High-power view of the lower part of the utricle showing two papillomatous lesions attached to local thickened areas of the wall. These are proliferative lesions containing both epithelial and mesenchymal elements as well as some multinucleated giant cells. These aggregates of cells probably represent focal lesions due to the measles virus similar to those occurring in the skin. The endolymph has increased affinity for the stain.

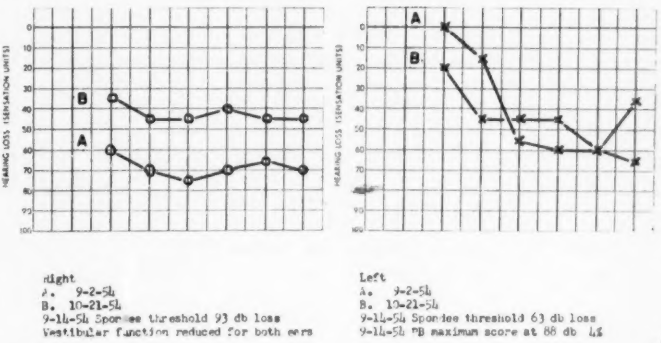


Fig. 4.—Age 66. Impairment of hearing and dizziness of sudden onset associated with meningoencephalitis.

of the blood stream, a complication of a viremia. The degenerative changes in the stria vascularis suggested that this was the route of entry to the cochlear duct. The vestibular part of the labyrinth was affected on one side only (Fig. 2). The pathologic process in these ears could best be designated as an "endolymphatic labyrinthitis" (Fig. 3).

The case presented by Nager [2] in 1907 showed somewhat different findings in that there had been a marked inflammatory reaction in the perilymphatic space with hydrops and degenerative changes in the basal coil only. The pathology in the inner ear in this case had been an extension from the subarachnoid space, and, since the child had recovered from the disease, it was probably an extension from a measles meningoencephalitis.

These two cases therefore demonstrate two routes by which the measles virus may invade the inner ear.

Another example of a virus which is capable of damaging the inner ear at an early stage of its fetal development is rubella. The available pathologic evidence suggests that the virus reaches the cochlea of the fetus by way of the circulation and affects the stria vascularis.[3,4] After the third month the development of the ear appears to be no longer susceptible to the virus.

Bilateral nerve-type hearing impairment and labyrinthine disturbance has been observed in one case in association with meningoencephalitis (Fig. 4) in which no infecting agent could be discovered. Hence an inflammatory reaction due to a virus seemed to be the probable etiology with a direct extension from the meninges to the labyrinths and secondary degenerative changes in the sense organs of the inner ear.

The association of herpes about the ear along with facial paralysis, and sometimes inner-ear deafness and loss of vestibular function, known as the Ramsay Hunt syndrome, is thought to be due to a neuritis or ganglionitis of viral etiology.

Another route by which a virus infection may invade or damage the inner ear is by way of the middle ear. Secretory otitis media, either acute or subacute, may be complicated by an inner ear complication with deafness and dizziness. Several examples have been observed.

Report of Cases

CASE 1.—A woman, age 32. First seen in 1946, with a history of discharge in right ear in childhood and again during the past two years. There was an attic cholesteatoma in the right side with an intact membrana propria. The left drum showed retraction of Shrapnell's membrane, but an intact drum. The hearing was depressed to the 15 db. level in both ears for speech frequencies. Tests indicated bilateral impairment of Eustachian tube function. Radium therapy to the tubal orifices produced no improvement.

A modified radical mastoidectomy was done on the right ear in August, 1947, resulting in a healed cavity with a pure-tone air-conduction threshold averaging 15 db. for speech frequencies.

On April 8, 1949, the patient was seen with a mild upper respiratory tract infection and mild acute nonsuppurative otitis media on the left side. A small crust was seen lodged in the depression in Shrapnell's membrane. The middle ear was filled with fluid, but the drum was translucent, with normal contour. Three weeks later the hearing became depressed for AC and BC sound, followed by severe dizziness. Cultures showed no definite pathologic organisms. Therapy with large doses of sulfadiazine, penicillin, and chlortetracycline Aureomycin was given for nine days. The hearing did not return in the left ear, and the cold caloric response remained absent. The crust came away from the Shrapnell's area, leaving a small pocket which remained dry.

During a subsequent attack of nonsuppurative otitis media an attic cholesteatoma became fully developed and in 1950 a modified radical operation was done, resulting in a dry healed cavity.

Comment: Damage to tubal function had occurred in both ears in childhood. This led to formation of an attic cholesteatoma on the right side. On the left side there was recurring nonsuppurative otitis media. During one episode of secretory otitis media with transparent fluid in the middle ear a rapid and permanent loss of inner ear function occurred. The etiology was apparently a virus infection. During a subsequent inflammatory episode a small attic cholesteatoma developed in the left ear also and required surgery.

The route of extension to the inner ear in this case appears to have been by direct spread from the middle ear.

Case 2.—Man, age 57. On the first examination at the University of Chicago Clinics, March 22, 1950, the complaint was as follows: 1. Severe deafness in left ear of five years' duration. Severe deafness in right ear for the past year. 2. Unable to understand speech adequately with the use of a hearing aid. 3. One severe dizzy spell had occurred five months earlier, followed by some improvement of hearing temporarily.

Hearing tests showed severe bilateral threshold loss by AC with positive Rinne tests. Both eardrums were thickened and opaque, with moderate retraction.

The impression given by several consultants had been that he had a hydrops of the labyrinth (Ménière's disease) on the left. Possibly also on the right. He had been advised to use a hearing aid. This was at first considered as the most probable diagnosis, and treatment was begun on that basis.

Histamine therapy was given intravenously daily for one month, then by subcutaneous injection for two months, one to three times a week.

Hearing showed gradual improvement, and treatment was stopped at two and one-half months. One month later he was reexamined. Although the left eardrum was thick, opaque, and retracted, the chalky appearance of the malleus suggested fluid in the middle ear. The hearing test showed the bone-conduction threshold to have improved, suggesting conduction deafness.

The eardrum was therefore incised and fluid evacuated by catheter inflation of the Eustachian tube.

The hearing improved further for both AC and BC (Fig. 5).

The fluid in the right middle ear kept recurring. Treatment included application of radium to the Eustachian tubal orifices. Hearing remained good; there were no dizzy spells, and after two years the fluid no longer collected in the ear. BC thresholds remained at about the AC level when adequate masking was used.

In April, 1952, two years after beginning of treatment, during an upper respiratory infection the hearing in the right ear was again suddenly impaired. After this incident the threshold at 2048 d. v., which had reached the 15 db. level throughout 1951, remained at the 60 db. level, with some elevation also at 1024 d. v. (Fig. 6).

In September, 1953, the hearing loss in the left ear had regressed further. The loss which had seemed to increase during upper respiratory infections now was permanent although variable from week to week.

A further course of I. V. histamine gave no improvement.

In 1956 the patient was again seen for the complaint of recurring dizzy spells, often causing him to fall. Hearing was still severely impaired in both ears. Various medical treatments have since been used with no definite improvement, although there is considerable fluctuation in the hearing from time to time.

The present diagnosis is hydrops of the labyrinth, right ear.

Comment: This case illustrates the effect a nonsuppurative middle-ear infection may have on the inner ear.

The course of events may be summarized as follows:

1. Probable bilateral tubal and middle-ear disease in childhood, which recurred in late adult life.

2. Inner-ear deafness, left ear, probably the result of middle-ear inflammation.

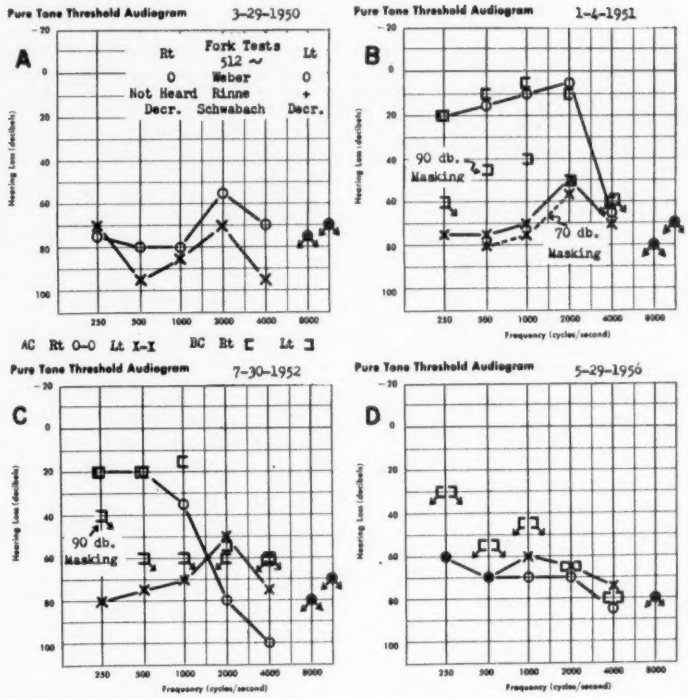


Fig. 5 (Case 2).—Man aged 57. (*A*) AC pure tone thresholds (3/29/50). Bone conduction decreased for 512 fork, bilateral. Marked deafness and dizzy spells for over one year. (*B*) AC and BC thresholds (1/4/51) after evacuation of fluid from right middle ear on repeated occasions. No further vertigo. (*C*) AC and BC pure tone thresholds (7/30/52). Marked hearing loss at 1000 and 2000 frequencies in right ear followed an acute upper respiratory infection. No dizziness. (*D*) Increase in the hearing loss on the right side for AC and BC pure tones. Recurring dizzy spells, deafness fluctuating in degree. Impression: Labyrinthine hydrops, right ear. Left ear essentially unchanged.

3. Inner-ear deafness and dizziness, right ear, secondary to chronic secretory otitis media, thought to be due to virus infection.

4. After recovery under therapy which was continued for two years, there was a sudden onset of partial inner-ear deafness, right ear, associated with an upper respiratory infection probably due to a virus.

5. During the following two years the classical picture of a labyrinthine hydrops developed in the right ear.

The history in this case seemed to show that a secretory otitis media which was probably due to virus infection had been responsible for the severe impairment of inner-ear function when first seen and for the subsequent recurrence of inner-ear deafness and later the classical picture of labyrinthine hydrops.

CASE 3.—During the acute stage of an upper respiratory infection, deafness, tinnitus and dizziness and some discomfort developed in one ear. Under the care of an otologist he was reported to have slight injection of the drum on the affected side. Cultures of the upper respiratory tract showed no pathogenic bacteria. Adequate antibiotic therapy was given without causing any change in symptoms. There was gradual recovery of equilibrium, but the hearing was permanently lost.

This case was interpreted as a labyrinthitis due to a virus infection which extended via the middle ear.

In addition to the cases cited above, which illustrate clinically and in some cases give pathologic evidence of at least three routes by which virus infections may invade and damage the inner ear, there are other instances in which the cause of a sudden deaf-

ness, with or without dizziness, may be a virus infection.

During the past 10 years four cases were seen which seem to fit into this category on the basis of the clinical history of an associated infection of the upper respiratory tract.

A typical history of this group was as follows: sudden onset of tinnitus, impaired hearing and moderately severe dizziness in one ear from one to two weeks after the onset of an acute upper respiratory infection; no evidence of suppurative disease in the upper respiratory tract. All four cases showed some improvement in hearing during the subsequent three months.

When considered in the light of the total number of cases of sudden deafness seen in the past 10 years, the number in which a virus infection might be considered as the probable etiology was relatively small. In the group occurring in middle life or after, some type of vascular lesion was considered to be the etiology in a much greater number.

In children the commonest cause was mumps. This had been sometimes overlooked until the child reached school age and was given a hearing test. If occurring during school years, the complaint of noises in the ear usually caused the parents to bring him for an examination.

Sudden deafness with or without vertigo is not an uncommon occurrence in the third decade of life without any demonstrable systemic or local disease that might be considered as the etiology. Many such cases have been under observation for many years after the incident occurred so that progressive disease seems to have been excluded as the etiology.

Summary

Sudden deafness with or without dizziness sometimes presents a diagnostic problem.

When such clearly differentiated conditions as bacterial labyrinthitis, tumor, trauma, drugs, and idiopathic hydrops (Ménière's disease) are excluded, three fairly large groups remain—virus infections, vascular lesions, and a third group, consisting mainly of young adults, in which the etiology is undetermined.

Virus infections which are best known as causes of deafness are mumps, measles, and maternal rubella. Less frequent syndromes which probably have a viral etiology include combinations of deafness with herpes oticus, meningoencephalitis, and neuritis. Virus infections of the upper respiratory tract occasionally invade the inner ear by way of the tube and middle ear.

Known pathways for virus infection to reach the inner ear are by way of the blood stream, the meninges, and the middle ear.

950 E. 59th St. (37).

REFERENCES

1. Lindsay, J. R., and Hemenway, W. G.: Inner Ear Pathology Due to Measles, Ann. Otol. Rhin. & Laryng. 63:754-771 (Sept.) 1954.

2. Nager, von F. R.: Beiträge zur Histologie der erworbenen, Taubstummheit, Zeitschr. Ohrenh. 54:217, 1907.

3. Nager, F. R.: Histologische Ohruntersuchungen bei Kindern nach mütterlicher Rubella, Pract. oto-rhino-laryng. 14:337-359, 1952.

4. Lindsay, J. R.; Carruthers, D. G.; Hemenway, W. G., and Harrison, M. S.: Inner Ear Pathology Following Maternal Rubella, Ann. Otol. Rhin. & Laryng. 62:1201 (Dec.) 1953.

Cancer of the Retromolar Area

A Study of Twenty-Eight Cases with the Presentation of a New Surgical Technique for Their Treatment

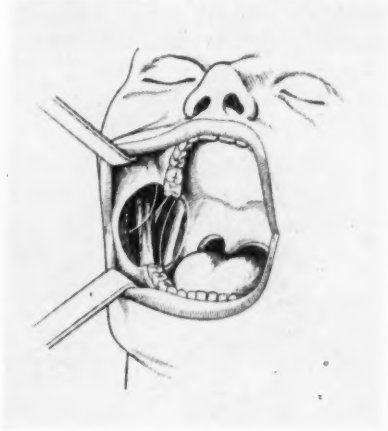
JORGE FAIRBANKS BARBOSA, M.D., Sao Paulo, Brazil

Anatomy

It is common to run across the term "retromolar region" in books and articles on cancerology. The term is used to designate the part of the buccal cavity which lies between the two dental arcades on either side and behind the roots of the last molar teeth, and which establishes a communication between the medial and vestibular portions of the buccal cavity when the dental arcades lie in contact. However such a definite anatomical "region" has no existence as far as could be vertified by an ample search of the literature. Accordingly, we shall call this part of the buccal cavity the retromolar "area" rather than "region," seeing that it lacks definite anatomical limits although it can always be identified as a locality. The "area" can also be identified as the "retromolar space," so called by Bertelli, being the portion joining the two alveolar borders, superior and inferior, as well as the parts of these borders that lie behind the roots of the last molar teeth. Externally the area passes imperceptibly to the corresponding internal aspect of the cheek and inwards it merges into the neighboring soft palate. In the majority of instances the internal limits are marked by a fold of mucosa caused by the prominence of the pterygomandibular ligament.

The retromolar mucosa is thick and attached to the skeleton at the superior maxillary tuberosity above and the posterior extremity of the inferior gingival border below. It is flaccid like the buccal mucosa, being greatly mobile and elastic at the angle joining the two alveolar arcades. It lies directly over the thin buccinator muscle which separates it from the buccal pad of fat. By following this pad we can easily visualize the deep structures of this area, which are well demonstrated in Figure 1 (modified from Sicher). Externally they form the anterior border of the ascending ramus, almost all of which is invested by the tendinous insertion of the temporalis muscle. Crossing this border is the buccal nerve. Internally lies the anterior edge of the internal pterygoid muscle. In the acute deep angle formed by the internal pterygoid and the ascending ramus, which is open superiorly, run the two branches of the maxillary nerve—the mandibular and the lingual. These three nerves—the buccal, mandibular, and lingual—are all sensitive and easily accessible to cancerous infiltra-

Fig. 1.—Demonstrating the deep structures of the retromolar area. (Modified from Sicher.)



Submitted for publication March 16, 1958.

Chief of the Head and Neck Service, Instituto Central of the Associacao Paulista de Combate ao Cancer.

tion from the retromolar area, explaining the painful symptomatology of these tumors.

The buccal pad of fat passes into the pterygomandibular angle, ascends to the zygomatic or infratemporal fossa, and occupies the free space between the pterygoid and temporal muscles; it surfaces at the lower limit of the temporal fossa enclosing the anterior border of the temporal muscle. It is along the weak fatty tissue of this pad of fat that the deeper layer of the area passes through the complicated labyrinthine planes of the zygomatic and pterygomaxillary fossae and to the inferior part of the temporal fossa. This fact explains the deep infiltration of retromolar tumors to reach and include the deep spaces of the tonsillar and prestyloid pharyngeal space, seeing that all these spaces intercommunicate amply.

Vessels and Nerves.—The retromolar mucosa receives a sensory supply from the trigeminal principally through the buccal nerve, while a limited portion of the floor of the mouth, anterior pillar, and tongue are innervated by the lingual nerve. According to Hollinshead, the roof of this area receives a sensory supply from the facial; yet there is no conclusive evidence of the existence of sensory fibers in this seventh cranial nerve.

The arterial supply comes principally from branches of the internal maxillary and facial arteries.

The veins are tributaries of the internal jugular and of neighboring venous plexuses, especially the internal pterygoid plexus.

Lymphatics pass to nodes in the submaxillary region and to the upper internal jugular chain.

Incidence and Etiology

Table 1 demonstrates the distribution of our 28 cases as regards age, sex, and color.

TABLE 1.—*Distribution of Cases by Age, Sex, and Color*

Age, Yr.	No.	Sex	No.	Color	No.
40 to 49	7	Males	26	White	26
50 to 59	8	Females	2	Negro	1
60 to 69	8			Yellow	1
70 to 79	5				

TABLE 2.—*Influence of Alcohol and Smoking on Etiology of Retromolar Tumors*

Alcohol	No. Cases	Smoking	No. Cases
—	7	—	0
+	4	+	8
++	3	++	6
+++	5	+++	7
++++	8	++++	6
No inquiry made	1	No inquiry made	1

* Cases graded + to ++++ as regards intensity of habit; addicted smokers and alcoholists are designated by +++ or ++++; — indicates absence of the habit.

While admitting that factors related to chronic irritation of the buccal mucosa can favor the occurrence of these tumors, yet we must realize that in this particular situation dental sepsis, broken stumps, and misfitting prostheses cannot have the same importance they have elsewhere in the buccal cavity. A particular exception, however, exists in the case of tumors arising from the alveolar margins and secondarily invading this area.

Alcohol and tobacco appear to us to play a definitely important role. Approximately 50% of the patients (13 out of 27 of whom a particular inquiry was made) were confirmed tobacco and alcohol addicts, that is, they smoked 30 or more cigarettes a day and drank concentrated alcoholic drinks such as *pinga* (a Brazilian national drink) more than three times daily. Many were addicted drunkards.

Only 1 case in 17 in which the serological reactions for syphilis were done proved to be positive, showing that syphilis has little importance in the etiology of these tumors.

To 25 queries as to cancerous antecedents in the blood relations we had four positive responses, i. e., practically the same relation that we have observed in other sections of the buccal cavity. In three cases the retromolar lesion was associated with another cancer, a finding which is similar to our observation in cancer of the pharynx and which seems to indicate the state of cancerous predisposition found in these patients.

TABLE 3.—*Duration of Complaint at the Time of Admission*

Duration, Mo.	No. Cases
1	3
2	4
3	5
4	2
5	1
6	3
7	2
8	5
9	1
12	2
Average: 5.1 mo.	

During our routine E. N. T. examination we have had the opportunity to observe papilliferous papillomata in the retromolar area and its neighborhood, commonest on the anterior pillar. At the same time some of the cancers in this area have the macroscopic appearance of papillomas, sufficiently similar in aspect to the benign papillomas to suggest the possible origin of some cases in benign papillomas.

Symptomatology

The average duration of patients' complaints at the time of their interview was 5.1 months (Table 3).

The advanced condition of the lesions at the time they presented, in spite of a short 5.1-month period of evolution, speaks in favor of a rapid progress—a finding which is understandable in view of the special situation of the tumor in a highly mobile area with weak cellular spaces in its depth facilitating infiltration. Similar characteristics, according to Rodrigues, explain the rapid evolution of cancers of the tongue.

Few cases develop silently; one of our patients presented himself to a dentist because of looseness of his last superior molar tooth on the right side; the dentist inadvertently extracted the tooth, noting meanwhile a whitish lesion in the retromolar area developing painlessly. Another patient observed enlargement of lymph glands in his neck, first noticing a smollen nodule on the same side as a later developing "ulcer in the mouth."

Generally these cancers are painful (23 of our 28 patients), the pain being usually intense but not incapacitating, and accentuated principally on opening the mouth, causing some difficulty in eating. The pain is continuous or intermittent and of a pricking character. It may be localized in the area (12 cases) or radiating (11 cases), mainly to the ear and ipsilateral dental arch. It is generally the pain which draws attention to the ulcer in the mouth; this was the case in 19 patients who had noticed the presence of a lesion at the time of their first interview.

As we have seen, the buccal pad of fat lying outside the buccinator, and in close relation to the mucosa, forms a weak cellular space, permitting easy access of the tumor to the masseteric, temporal, and prestyloid spaces of the zygomatic region. This facilitates infiltration of the muscles of mastication, principally the masseter and internal pterygoid, and as a consequence gives rise to trismus, which was observed in 10 of our cases. Infiltration of the masseter may give an externally visible swelling.

Trismus and pain on movement of the mandible hamper feeding. Ten of our patients gave a history of marked loss of weight when admitted. Table 4 gives, in order of decreasing frequency, the complaints of our patients. Only 2 had observed swollen cervical glands on admission; we

Fig. 2.—Ulcerovegetating papillary tumor of the retromolar area.

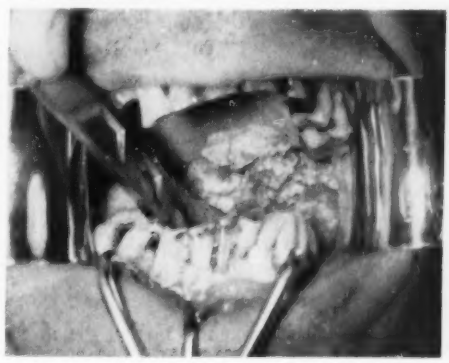


TABLE 4.—*Principal Complaints on Admission in Order of Decreasing Frequency*

Complaint	No. Cases
Pain in the retromolar area: local, no radiation (12) / Same pain plus radiation (11)	23
Presence of an "ulcer" in the retromolar area	19
Trismus	10
Loss of weight	10
Excessive salivation	4
Swelling of face	3
Hemorrhage	2
Cervical glands	2
Halitosis	1
Swelling of tongue	1
Pain in the throat	1

shall see further on that 17 others had evidence of lymphatic metastasis on clinical examination, and 5 more were suspicious. Six cases had no glandular involvement.

Microscopical Pathology

All cases were subjected to biopsy. With the exception of one case of transitional-cell carcinoma and another of adenocarcinoma, all our cases were stratified squamous epitheliomata.

Macroscopic Pathology

A. Primary Lesions.—Our series is too small to permit classification; we can only comment on the principal forms of lesion we have had the opportunity to observe.

In one of our cases which had received intensive irradiation recurrence took the form of a superficial ulceration with a clean reddish floor and defined borders of an irregular ameboid or serpiginous nature involving all the retromolar area and extending on the buccal aspect of the anterior pillar.

In most instances (16 cases) the lesions were ulcerodestructive and associated with marked infiltration at their margin (Fig. 3). Such lesions exhibit marked tissue destruction and ulceration passing posterosuperiorly to the posterior extremity of the superior gingivobuccal groove. Sometimes such lesions assumed the appearance of a depression or sulcus of the depth of the groove cutting through vegetating neoplastic masses; in other cases the appearance was frankly

Fig. 3.—Ulcerodestructive lesion of the right retromolar area.

crateriform. The margins sprouted as fleshy coarse granulations, easily bleeding, and the depths were covered with necrotic material bleeding through vegetating tumor tissue. It is this type of tumor that is usually associated with intense infiltration of the neighborhood and accompanied by trismus.

Eleven cases were of the ulcerovegetative type; two of these were frankly papilliferous (Fig. 2) and whitish on the surface, denoting a high grade of cornification; in two others the ulcers vegetated with gross fungating friable granulations reminiscent of cauliflower vegetation. In the remaining seven ulcers the fleshy vegetations were less pronounced centrally but were developed at the margins, resembling somewhat the granulations of tissue healing; these cases were also associated with accentuated infiltration.

Direction of Propagation: Propagation passed in a superficial as well as in a deep direction. Superficial extension occurred in all directions, with no apparent special predilection, invading the posterior part of the gingival border, the buccal aspect of the

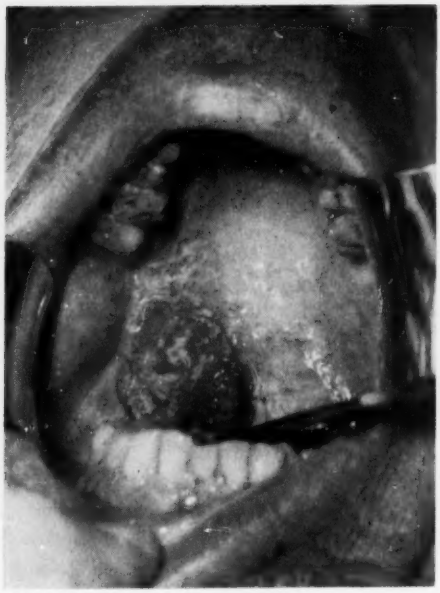


Fig. 4.—Ulcerovegetative lesion of the right retromolar area.

cheek, anterior pillar, its neighboring soft palate, the posterior part of the floor of the mouth, the tonsillar region, and the neighborhood of the base of the tongue. It is our impression that the posterior part of the inferior gingival border was the part most commonly involved. The tendency to early spread is demonstrated by the fact that only two of our cases were localized within the retromolar area.

Deep extension of the tumor passes through the buccal pad of fat to the masseteric region, giving a visible external swelling; likewise along this pad tumors spread to the prestyloid portion of the zygomatic fossa—paratonsillar region—and from there along the superior constrictor they spread and invade the tonsillar region. However, it seems to us that the tonsillar region is more easily invaded by superficial spread across the anterior pillar.

B. Metastases.—In 17 of our 28 cases (60%) there was clinical evidence of glandular metastasis at the time of admission, while in 5 others there was clinical suspicion of such involvement (Table 5).

TABLE 5.—*Clinical Involvement of Cervical Glands in Twenty-Eight Cases*

Glandular Involvement	No. Cases
Ipsilateral	12
Bilateral	5
Suspected	5
No	6

The glands involved were the superior jugular and the submaxillary.

Table 6 correlates clinical and histological findings in 10 cases in which a radical neck dissection was done.

Routine histological examination, like clinical examination, is falsely negative in some cases unless serial sections are made in all glands removed, which is impracticable. By serial sections in cases already reported negative to routine histology, Ogura and Belo demonstrated 40% involvement of glands. This fact cited by Nicelli in discussing metastatic involvement in cancer of the larynx should also apply to retromolar cancers. Nevertheless, examination of our surgical specimens showed us that in some cases simple inflammatory enlargement of glands is misleading and mistaken for metastases so that actual metastatic involvement is not as common as one is led to believe by clinical findings.

Treatment

Because of inaccessibility, great mobility, and intimate skeletal relations, the retromolar area offers poor prospects for irradiation; facility of infiltration is commonly evidenced by osseous destruction, and for

TABLE 6.—*Glandular Involvement*

	No. Cases
Evidence of clinical glandular involvement and histology negative	3
Evidence of clinical glandular involvement and histology positive	3
No evidence of clinical glandular involvement and histology negative	2
Evidence of clinical glandular involvement suspected and histology negative	1
No histological report available	1

this reason radiation therapy has little or no effect against these tumors. Eleven of our cases received an initial radiation treatment as the only possible treatment. The results (Table 7) confirm the claims we have forwarded: of the eleven cases two are still under treatment; one has been lost track of, and all the rest were uncontrolled by radiotherapy.

On the other hand, surgical treatment offers sufficiently encouraging results. The technique, which we will describe further on, aims at removing, together with the retromolar area, all the neighboring regions customarily invaded by the tumor (masseteric region, prestyloid part of zygomatic fossa, superior maxillary tuberosity, ascending ramus of the mandible, and the posterior part of the horizontal ramus). These are removed in one block, together with all the structures removed by a radical cervical block dissection of the same side, or even of both sides if clinical evidence points to bilateral involvement.

We are not in favor of a prophylatic block dissection; however, we agree with Martin that where removal of the primary lesion necessitates an approach through the neck, we should then profit by this opportunity to do a block dissection. We hold this view for three reasons: first, because we can never guarantee against tumor contamination of the neck field; secondly, our incision, by interrupting the natural routes of lymphatic drainage, may deviate such drainage to new inaccessible areas; thirdly, because the resulting operative scars make a later block dissection difficult when it becomes necessary.

TABLE 7.—*Results of Radiotherapy in Eleven Cases of Retromolar Cancer*

A. Cases treated exclusively by irradiation		8
Dead of cancer	5	
Lesion uncontrolled 2.5 mo. after treatment	1	
Still under treatment	1	
Lost contact	1	
B. Initially irradiated as a curative treatment and subsequently operated on because the primary lesion could not be controlled		2
Asymptomatic 21 months after complementary surgical treatment	1	
Dead	1	
C. Cases initially irradiated as a curative treatment, subsequently operated on because the primary lesion could not be controlled, and then once more irradiated (radium) for new recurrence		1 *

* Still under observation.

TABLE 8.—*Results of Surgical Treatment in Ten Cases of Cancer of Retromolar Area*

A. Cases receiving surgical treatment only		4
Asymptomatic for 6 mo.	1	
Asymptomatic for 10 mo.	1	
Asymptomatic for 15 mo.	1	
Died *	1	
B. Surgery following recurrence after radiotherapy		2
Asymptomatic for 20 mo.	1	
Died †	1	
C. Surgery followed by prophylactic irradiation		3
Asymptomatic for 4 mo.	1	
Asymptomatic for 5 mo.	1	
Still under treatment	1	
D. Surgery for recurrence after radiotherapy in turn followed by radium implantation because of recurrence (still under observation)		1

* Died of a recurrence different histologically during a series of plastic interventions.

† Postoperative complication.

Ten of our cases were treated surgically with the object of a radical cure; two of them had already been treated by irradiation; two others received postoperative radiotherapy prophylactically against possible recurrence. Table 8 demonstrates the results.

In conclusion, of 10 cases treated surgically 6 are asymptomatic (4, 5, 6, 10, 15, and 21 months respectively); 2 are still under treatment, and 2 died. Of the fatal cases one died on the 11th postoperative day of a toxic-infective collapse with bronchopneumonia; the other died 8 months later during the course of a series of plastic surgical operations for the closure of a gap in the cheek; this patient died of an operative complication, and at autopsy a tumor was detected in the retromolar area (malignant mesenchymoma) of a different histological nature from the original carcinoma. We cannot tell whether the repeated surgical trauma in zone already predisposed to malignancy could be responsible for such a recurrence.

Surgical Technique

The operation is performed with the patient on his back and his head turned to the

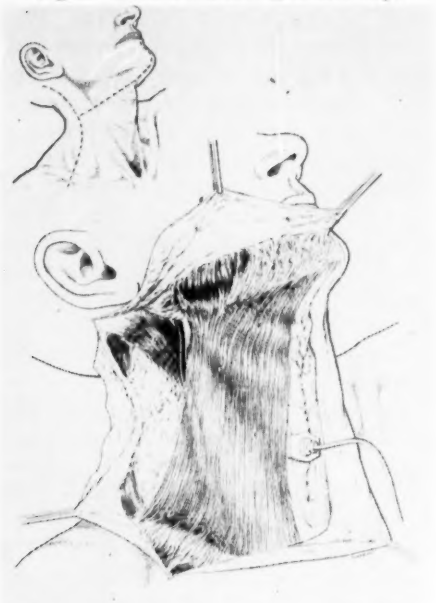
healthy side. A low subisthmic tracheotomy may be performed at the beginning or immediately on ending the operation. Potentiated anesthesia is used.

The operation may be divided into two parts. The first part, carried out in five successive steps, is in reality a radical cervical block dissection. We have described our technique for cervical block dissection in another article * and shall not repeat the description here. The points we should like to emphasize, however, are the following:

We prefer to carry the dissection from behind forward, facilitating exposure of the jugular vein, and structures related to it, from its deep aspect, thus enabling us to follow the phrenic nerve to its point of origin from the branchial and cervical plexuses. At the same time a large number of lymphatic channels are exposed and ligatured, and, in particular on the left side, the thoracic duct can be identified and ligatured, avoiding an embarrassing postoperative chylorrhea.

* Barbosa, J. F.: Radical Laryngectomy with Bilateral Neck Dissection in Continuity, A. M. A. Arch. Otolaryng. 63:372-383, 1956.

Fig. 5.—Incision and raising of neck flaps.



Again we insist on adequate cleaning of the subtrapezial space on the undersurface of the trapezius, where we secure a pedicle formed by the accessory nerve and the transverse cervical artery.

The successive steps in the cervical block dissection are the following:

I. Incision and raising of neck flaps (Fig. 5).

II. Dissection of the posterior triangle (Fig. 6).

III. Section of the inferior end of the sternocleidomastoid muscle. Ligature and section of the inferior end of the internal jugular vein (Fig. 6).

IV. Dissection of the carotid artery (Fig. 7).

V. Section of the inferior pole of the parotid gland and the posterior belly of the digastric. Ligature and section of the upper end of the internal jugular (Fig. 7).

VI. Deep dissection of submaxillary and submental regions (Fig. 7).

Fig. 6.—(Upper left) Dissection of the posterior triangle. (Upper right) Section of the inferior end of the sternocleidomastoid muscle. (Lower) Ligature and section of the inferior end of the internal jugular vein.

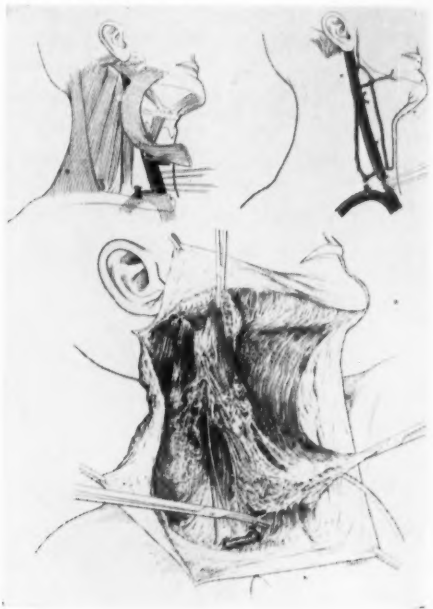


Fig. 7.—Dissection of the carotid artery. Section of the inferior pole of the parotid gland and posterior belly of the digastric. Ligature and section of the upper end of the internal jugular vein. Deep dissection of the submaxillary and submental regions, including the anterior belly of the digastric muscle.

Fig. 8.—The whole surgical specimen is now free except for its attachment to the inferior border of the mandible. (More detailed description of these different steps of neck dissection is found in another paper published by us under the title "Radical Laryngectomy with Bilateral Neck Dissection in Continuity," A. M. A. ARCH. OTOLARYNG. 63:372-383, 1956.)

At the end of this part of the operation the whole surgical specimen is free except for its attachment to the inferior border of the mandible, as shown in Figures 8 and 9. The next step is to excise the primary lesion.

Fig. 9.—As in Figure 8, we see the whole surgical specimen attached to the inferior border of the mandible.

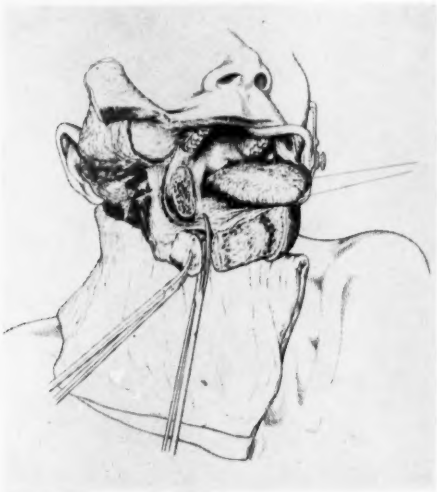
VII. Section of the lower lip, the vestibular mucosa, and masseter muscle. The facial pedicle is ligatured close above the submaxillary gland at the anterior border of the masseter; at this height the artery is crossed by the mandibular branch of the facial nerve which runs parallel to the inferior border of the mandible. This branch should be identified and preserved to avoid paralysis for the external angle of the lower lip. The upper flap is now elevated to the level of the zygomatic arch, and the parotid gland is isolated from the posterior border of the mandible and partially raised with the flap, with avoidance at the same time of injury to the fibers of the facial nerve which traverse the gland and are included in the thickness of the superior flap. The lower lip is cut through its whole thickness in the middle line, and the mouth forcibly opened with a mouth gag, permitting ample

visualization of the retromolar tumor. With scissors the soft parts are sectioned along the inferior vestibular groove, the buccal mucosa being cut, together with underlying buccinator, passing posteriorly till the external aspect of the superior gingival margin is reached behind the last molar, or more anteriorly if it is necessary to secure a greater safety margin. The mucosal resection should pass at least 2 cm. away from the limits of the tumor. Finally, the masseter is cut horizontally, with the electric knife, below the zygomatic arch. Now is the time to liberate the coronoid tubercle of the mandible from the tendinous intersection of the temporal muscle.

At the end of this stage of the operation the horizontal ramus of the mandible is amply visualized.

VIII. Section of the mandible, floor of mouth, and palate. The mandibular periosteum is elevated at the level of bone section, which varies according to the size of the tumor. In extensively invasive tumors it is usual to section the bone at the level of exit of the mental nerve, so as to include in the resection all the length of the mandibular nerve, which forms a selective pathway for neoplastic growth. Generally, however, the section can be done more posteriorly in the region of the molar teeth. A Gigli saw is usually used for this section.

Fig. 10.—The content of radical neck dissection attached to the inferior border of the mandible is under the compress. The lower lip was sectioned in the midline. The buccal mucosa and buccinator muscle are cut, passing at least 2 cm. away from the tumor. The upper flap was raised with the parotid gland, in order to protect the facial nerve, till the zygomatic arch was reached. The masseter is sectioned at this level. The mandible is sectioned, and we begin the section of the floor of the mouth with snips of the scissors.



The tip of the tongue is pulled over to the side away from the operative field; the posterior part of the mandible is pulled outwards, thus raising the floor of the mouth. With snips of the scissors the mucosa and underlying soft parts are cut (Fig. 10), including some posterior fibers of mylohyoid and lingual nerve, and sometimes the sublingual gland and sublingual artery as well. Proceeding backward, the line of section is carried to the anterior pillar at its insertion in the tongue, passes along the glossoamygdaloid sulcus, to the posterior pillar, and up to the soft palate. In the soft palate the incision cuts mucosa, submucosa, a glandular layer, and tendinous fibers of the tensor palati. The levator palati should be avoided, if possible, to prevent a transpalatine communication between mouth and nasopharynx. The palatine incision continues until it meets the first incision in the vestibular portion of the mouth at the posterior extremity of the superior alveolar border. The incision should nowhere be nearer than 2 cm. to the limits of the tumor, and therefore it varies with the dimensions of the lesions. Frequently we meet cases invading the margin or base of the tongue, and in such cases the incision deviates, passing over the tongue. Frequently we have had to cut transversely across the tongue to the median raphe, pass along this raphe till the floor of the valecula, and along this floor to the pharyngoepiglottic fold to reach the posterior pillar. This permits inclusion, in the surgical specimen, of all the parts suspected of neoplastic contamination.

As we go deeper with the incision, the posterior part of the mandible is steadily

Fig. 11.—Appearance of the operative field on removal of the surgical specimen. The whole neck is cleared, the digastric cut along its extension. The muscles attached to the hyoid are the stylohyoid and styloglossus. Superiorly are seen the lines of section of the masseter and internal pterygoid and between them the lateral pterygoid. The muscle seen in the soft palate is the levator palati.

pulled outward, permitting better visualization and illumination of the operative field.

The posterior portion of the superior alveolar margin (maxillary tuberosity) is easily removed by chisel and hammer, together with the surgical specimen. If the tumor invades a large portion of the superior alveolar margin, all that portion can be removed by chiseling backward and included in the monobloc excision.

IX. Section of the medial pterygoid and disarticulation of the mandible. Forcible traction posterolaterally on the posterior part of the mandible permits easy visualization of the internal face of the ascending ramus; this face is covered by the internal pterygoid the fibers of which are under traction and separated from important neurovascular structures, including the carotid, which pass in the depth of the zygomatic fossa. The fibers can be safely cut with the electric knife. The muscle should be cut as high as possible, practically at the lower border of the lateral pterygoid. Together with the medial pterygoid, the lingual and mandibular nerves are cut, lying between the muscle and mandible. At the same time fibrous ligaments fixing this part of the bone are cut.

The whole surgical specimen now hangs by the temporomandibular articulation; by twisting it posterolaterally disarticulation is easily achieved, the whole specimen being removed in one block. Bleeding commonly occurs near the glenoid cavity because of rupture of fibers of the external pterygoid and the pterygoid plexus; this is easily ar-

Fig. 12.—Showing the limits of surgical resection. The surgical specimen removed en bloc contains all the structures usually removed in a cervical block dissection, a portion of the posterior mandibular ramus and all the ascending ramus, the soft parts of the retromolar area, nearly all the masseter and internal pterygoid, all the tonsillar region, and all the prestyloid pharyngeal space.

rested by a 00 absorbable surgical suture transfixing the muscle above the glenoid cavity.

X. Reconstruction. This is achieved by means of interrupted 00 chromic absorbable surgical sutures uniting the two lines of section of the buccal mucosa (Fig. 12) to the level of the red margin of the lip. Approximation is not possible in that part of the field corresponding to the retromolar area. Here the mucosa should be sutured to the muscular planes which form the depth of the gap in a way so as to completely close any communication between the buccal cavity and the neck. This raw area can be covered by a free skin graft; we have always found it easily epithelialized within the first 10-15 postoperative days just as in the case of ordinary tonsillectomy operations. Covering the first layer a second interrupted suture layer of 00 absorbable surgical suture or fine cotton approximates the muscular planes. This not only reinforces the first layer but also diminishes dead spaces. At the end of this suture one or two Penrose drains are left in the cervical field after a careful washing of this field with saline and then a penicillin solution. In our last cases we have used the technique of continuous aspiration with a very good result. The skin is closed with separate cotton sutures. A compression dressing is applied to the neck, and a gastric tube is introduced for feeding.

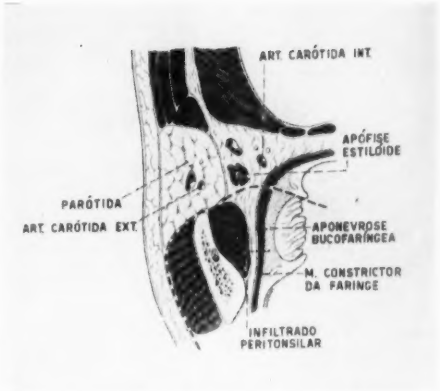


Fig. 13.—Horizontal section passing through the retromolar area. The interrupted line demonstrates the limits of resection. It is seen that they include the ascending ramus of the mandible covered by the masseter externally and the medial pterygoid internally. The whole tonsillar region and the prestyloid part of the zygomatic fossa are included.

Postoperative Care

On the first postoperative day the patient is immobilized in bed with intravenous alimentation and antibiotics. On the second day the patient is usually up, and tube feeding is started. Antibiotics continue until

Fig. 14.—The surgical specimen of the patient seen in Figure 2.

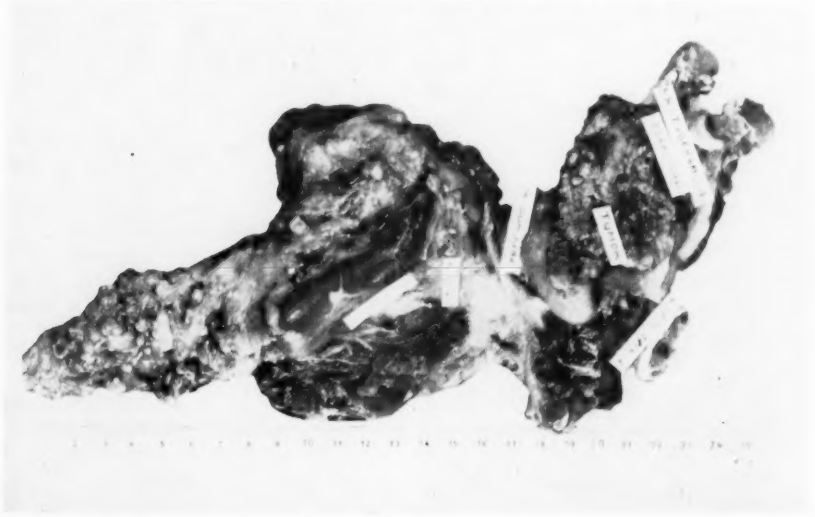


Fig. 15.—Postoperative aspects of a patient.

there is no fever. On the third day the dressing is changed, and drains are partly withdrawn. On the fourth day the drains are completely removed, unless there is suppuration, which is uncommon in these cases. On the fifth day some stitches are removed, and ordinary oral feeding started. All through these postoperative days the respiratory system should be aspirated several times a day by a tube introduced along the tracheotomy. Once secretion has disappeared, the tracheal cannula is removed and the tracheotomy allowed to close.

If pathological examination reveals lymphatic involvement, the patient is sent for postoperative irradiation once the skin incisions have healed.

Summary

The anatomy of the retromolar area is described with a view, principally, to explain the painful symptomatology of retromolar tumors, and the extensive infiltration of the masseteric region, the zygomatic fossa, and the tonsillar region.

This is followed by a clinical study of 28 cases of cancer in the retromolar area, with discussion of incidence, etiology, pathology, histology, and clinical evolution of these tumors.

Little can be done for these tumors by irradiation. A special technique for their treatment is presented, permitting their removal with a big safety margin. Analysis of early results that we have had are encouraging.

Rua Marconi 34-30.

BIBLIOGRAPHIES

Bertelli, D.: Contributi alla morfologia della mandibola umana con accenni alla insercioni dei musculi temporale e massetere ed alle origini dei musculi buccinatori e miloioideo, Arch. ital. anat. e embriol. 13:284-324, 1914-1915.

Hollinshead, W. H.: Anatomy for Surgeons: Vol. 1: The Head and Neck, New York, Paul B. Hoeber, Inc. (medical book department of Harper & Brothers), 1954.

Martin, H.; Del Valle, B.; Ehrlich, H., and Cahan, W. G.: Neck Dissection, Cancer 4: 441-499, 1951.

Nicelli, F.: Il problema anatomo-clínico delle Metastasi latero-cervicali del cancro laringeo, Arch. ital. otol. (Supp. 14) 64:38-53, 1953.

Rodrigues, A.: Os esvaziamentos ganglionares complementares: Estudo crítico, indicações e técnica, Arq. pat. 14:71-120, 1942.

Sicher, H.: Oral Anatomy, St. Louis, C. V. Mosby Company, 1949, p. 418.

Congenital Micrognathia

CHESTER M. WESEMAN, M.D., Berkeley, Calif.

Introduction

Pierre Robin,[25] in 1923, published a paper describing a syndrome encountered in the newborn, consisting of hypoplasia of the mandible, glossoptosis, and consequent respiratory and feeding difficulty. The anatomy pertinent to this syndrome is seen in Figure 1, which illustrates the attachment of the genioglossus muscle to the symphysis of the mandible. Shortening of the mandibular arch permits the tongue to fall back and thereby reduce the size of the hypopharynx. This condition is known as the Pierre Robin syndrome, and is referred to variously as "micrognathia," "micrognathy," "hypoplasia of the mandible," "receding chin and glossoptosis," "micrognathia with obstruction," and "ateliosis of the mandible."

Respiratory obstruction is the most important feature of this syndrome. If the obstruction is not satisfactorily relieved by placing the patient on his side or face, or if feeding is accompanied by repeated episodes of aspiration, the patient is in grave danger. It is the aim of this paper to emphasize that danger, which is a problem of the newborn and young infant, to review the various methods of management, and to urge positive and realistic treatment for the relief of the respiratory obstruction. The problems, mainly dental, of the older micrognathic child will not be discussed.

Embryology

The mandible is formed from the first branchial arch, which divides into the maxillary and mandibular processes. According to Keibel and Mall [14] its growth center appears as early as the 39th day and by the 42d day the ramus and alveolar process may be distinguished. The beginning of the coronoid process and the condyle becomes visible by the 55th day. By the middle of the third month the mandible has attained its characteristic shape. At birth

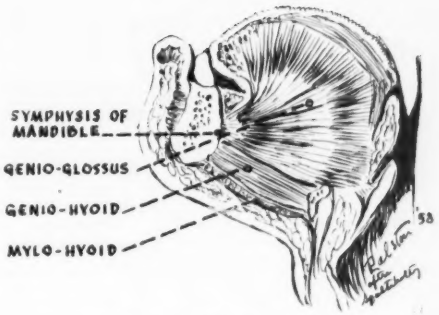


Fig. 1.—Median section through the floor of the mouth showing the attachment of the genioglossus muscle to the symphysis of the mandible.

Fig. 2. (after Retzius, in Keibel and Mall: "Human Embryology," J. B. Lippincott Company).—Head of a fetus of 117 mm. (approx. 4 months) in profile.

Submitted for publication April 4, 1958.

From the Department of Otolaryngology, Children's Hospital of the East Bay, Oakland, Calif.

Presented as a candidate's thesis in partial fulfillment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Inc.

Figure 3

Fig. 3 (after Scammon, in Arey: "Developmental Anatomy," W. B. Saunders Company).—Profiles, illustrating the changes in the form and proportions of the face throughout the life span.

Figure 4

Fig. 4 (after Retzius, in Keibel and Mall: "Human Embryology," J. B. Lippincott Company).—Head of a fetus of 25 mm. (approx. 2 months) in profile.

the lower jaw consists of lateral parts united at the symphysis by fibrous tissue. Osseous union takes place in the first or second year after birth.

During the first half of the third month the upper and lower lips of the fetus project about equally. The upper lip then begins to grow more rapidly so that it projects markedly beyond the lower lip, producing a physiological micrognathia during the fourth and fifth month (Fig. 2). This condition is overcome, in turn, by a more rapid period of growth of the lower jaw from the sixth to the ninth month.

Orban [23] further delineates this alternating growth rate of the upper and lower jaws (Fig. 3). He states that the growth of the mandible is at first small compared with that of the maxilla. The fetus of 2 or 3 months still shows a physiological micrognathia (Fig. 4), at which time the palatine processes are vertical and the tongue is in the nose in contact with the inferior edge of the septum (Fig. 5*A*). Between this stage and the time of complete union of the palate, at the end of the fourth month, a spurt in growth of the mandible (in length and width) takes place. This pulls the tongue

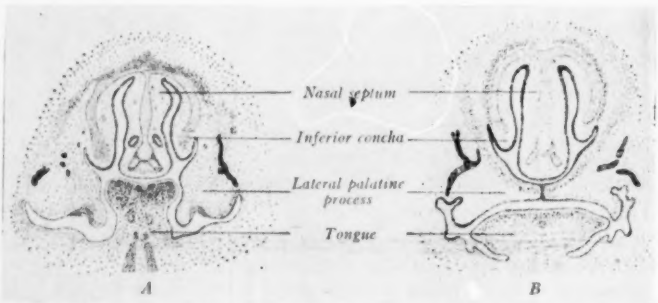


Fig. 5.—*A* (after Keibel in Arey: "Developmental Anatomy," W. B. Saunders Company), frontal section through the head of a human embryo of 8 weeks. The tongue is high and narrow between the vertical palatine processes. *B* (after Kallius in Arey: "Developmental Anatomy," W. B Saunders Company), frontal section through the head of a human embryo of 10 weeks. The tongue has evacuated the space between the palatine processes, and lies flat and wide within the mandibular arch. The horizontal palatine processes have fused with each other and will soon fuse with the nasal septum.

Fig. 6 (after Retzius, in Keibel and Mall: "Human Embryology," J. B. Lippincott Company.—Head of a fetus of 42.5 mm. (approx. 2½ months), in profile.

down and forward, out of the nose, permitting the transposition of the palatine processes to a horizontal plane, thereby fostering normal medialward growth and eventual union (Fig. 5*B*). This sudden growth of the mandibular arch, to the extent of producing mandibular protrusion (macrognathia) can be observed in the 10-week (42 mm.) embryo (Fig. 6). After this stage the maxilla again outgrows the mandible, producing another physiological micrognathia in the fourth and fifth month (Fig. 2), which is, as already stated, compensated by an increase in the growth of the mandible during the sixth to ninth months.

Normal Growth

The principal growth center of the mandible is in the condyle. Brodie,[2] who has written extensively regarding the growth of this structure, states that growth begins at a center and spreads toward the periphery, and that the rate is most intense at the center, diminishing peripherally; and, further, that the rate is highest when the cells begin their activity, and slowly diminishes. In a later article[3] the same author states that the condylar growth center appears in the 50 mm. embryo, displays its peak activity during the prenatal period, and persists to the 20th year. This condylar center is responsible for downward and forward development of the mandible. The growth is epiphyseal in character.

There is some controversy as to whether further growth of the mandible is appositional or interstitial, or both. Scott[29] states the points of common ground in this question. In so doing he defines growth as an increase in bulk; development, as an adaptation of that bulk to function.

1. The shape and general size of the mandible are genetically determined and are influenced by both intrinsic and extrinsic factors.

2. The extrinsic factors are developmental and functional in nature.

3. There is growth in the horizontal, vertical, and transverse directions, which is accompanied by adaptive adjustments in the same planes.

4. The mandible, like all other bones, is constantly changing in its adaptive capacity throughout the life of the individual.

His animal experimentation led him to conclude that interstitial growth possibly takes place. He demonstrated appositional growth on the posterior border of the ramus and the inferior border of the body of the mandible. He also demonstrated that the greatest amount of growth takes place in a region in which the most powerful of the muscles of mastication have their attachment. He quotes Thoma, who, as the result of animal experimentation, propounded three theories regarding factors which augment inherent growth tendencies.

1. The development of the jaws is due principally to occlusal force during mastication.

2. The development of the jaws is governed principally by the development of the teeth.

3. The development of the jaws is fostered principally by muscular activity.

Brodie[2] states, in regard to mandibular growth, that the greatest increment of anterior-posterior growth is at the posterior border of the ramus and results in a pushing forward of the entire bone. However, absorption trims the anterior, or coronoid, border back, only slightly less rapidly than deposition is occurring behind, thus permitting the ramus to grow slowly in this dimension. After the initial generalized period of growth has passed, increase in the height

of the body of the bone is almost entirely at the alveolar border, very little at the lower border [contrary to Scott's findings]. There seems to be no controversy regarding the mechanism of growth of the ramus; absorption of the anterior border makes room for the molar teeth, while the more rapid deposition of bone on the posterior border increases its bulk.

Abnormal Growth

As pointed out by many authors, interference with any of its growth centers will result in eventual deformity of the mandible. Brodie,[3] in writing of micromandibular development, states that it always is associated with other congenital anomalies, and that it is due to repression of the growth center of the head of the condyle. He further states that the days of fetal repression will never be made up, but that, once recovery has occurred, "everything goes along in the usual manner."

Keibel and Mall [14] state: "By a kind of inhibition process the early fetal arrangement may be retained in the adult to a marked degree." Gladstone and Wakeley [12] conclude that antenatal deformities, such as cleft palate, hare lip, agnathia, and cyclops, are usually the result of imperfect nutrition, or malnutrition of the embryo. They further state that direct inheritance of antenatal deformities seems to be the exception rather than the rule.

Davis and Dunn [6] mention imperfections in development of the first branchial arch as being responsible for this anomaly, and state that cleft palate is the most commonly associated malformation. Their concept of the embryologic mechanism is quoted:

> Considering the position of the child in utero, where the weight of the entire fetal body may be readily thrown on the vertex, it is easy to conceive how pressure of the sternal region in contact with the mandible might force the forming jaws together. If this undue force occurred at a time when the lower jaw was united and the upper jaw ununited, it is easy to understand how pressure on the chin could force the partly calcified lower jaw backward, straighten [sic] the obtuse angle to almost a right angle and prevent union of the horizontal plates causing a cleft in the soft tissues of the palate without involvement of the alveolar ridge or lip.

Sarnat and Gans [27] state:

> The underdeveloped mandible, like the overdeveloped one, is usually caused by a disturbance of the condylar growth center, congenital deficiency of, or injury to the growth center as the result of trauma, radiation, or inflammation.

Kiskadden and Dietrich [15] simply state "the reasons are unknown."

Pruzansky and Richmond [24] theorize that during embryonic development the tongue fails to sink below the palatal processes, because of the persistent micrognathia, and hence prevents their fusion. They cite the high incidence of micrognathia in a population of clefts involving only the hard and soft palate.

McCoy [21] implies that the dwarfed mandible does not correct itself spontaneously to the point of its hereditary and environmental potential. Beers and Pruzansky [1] studied head growth, using cephalometric roentgenography, in a typical case of congenital micrognathia with cleft palate, on which the Beverly Douglas operation had been performed. They found the pattern of neurocranial growth consistent with normal. Serial changes in the facial profile steadily improved by the reduction of the convexity of the profile as the mandible moved downward and forward. They observed that the most marked change took place during the first few months.

Warkany [32,33] has been able to produce congenital malformations in animal experimentation by bringing about a state of nutritional deficiency in the pregnant mothers. When a diet completely deficient in riboflavin was used, one-third of the young had anomalies which altered the external appearance; for example, short mandibles and protruding tongues. Of these abnormal young 44% had clefts of the palate.

Review of the Literature

The first definitive work to be published on this subject was that of Pierre Robin,[25] whose name has been given to the syndrome. He described the infant with a receding chin

and backward lowering of the root of the tongue who suffered from respiratory disturbance. He wrote again,[26] for the American literature in 1934, in which article he reviewed the subject and stressed positioning and "orthostatic feeding" in the management of these cases.

In 1925 Lenstrup[17] described two cases of typical "bird-face" (*Vogelgesicht*) infants with cleft palate, who were managed by positioning and careful feeding. In the author's words, by "constant careful attention they were successful in getting the children to thrive, and overcome the choking fits." He referred to Shukowsky's two cases, described in 1911 as "stridor congenitus," which were identical with his own cases. Kiskadden and Dietrich[15] quoted Shukowsky as recognizing the tongue as the offending organ in his first case in 1902, and suturing it forward to the lower lip. Lenstrup went on to state that cleft palate is found in all the cases, but that it is "scarcely of significance in the facies morbi." He further states "the somewhat drastic . . . therapy used, of pulling the tongue forward by means of sutures, or suturing it to the lower jaw, is scarcely to be recommended."

A reference in the otolaryngology literature appeared in 1930. Janse[13] described an 8-week-old infant extremely malnourished, with inspiratory stridor, a receding chin, a wide cleft of the hard palate, and marked retraction of the sternum with each inspiration. It was discovered that pulling the tongue forward gave relief, so a suture was placed through the tongue, run over a pulley and attached to a specimen bottle which was filled with varying amounts of water to regulate the degree of traction. The sutures pulled out on the third day, so a tracheotomy was performed. The patient died of bronchopneumonia five days later. Janse, though aware of Robin's work, emphasized "macroglossia" as the primary defect.

Eley and Farber[10] reported four cases of hypoplasia of the mandible in 1930. Each presented the typical picture of attacks of cyanosis and feeding difficulty, and each case had a cleft palate. One case was treated by emergency tracheotomy two hours after admission but died nine days later. In two others postural treatment was sufficient, and the third was managed additionally by a head brace which forced the mandibular rami forward. The authors stated that "surgical procedures such as suturing the tip of the tongue to the lip and tracheotomy, may be used but in our experience such measures were not necessary."

A device which is frequently tried in the management of micrognathia was described by Davis and Dunn[6] in 1933. This is a lip guard which attaches to a nursing bottle and which by pressure of the guard on the patient's upper lip forces him to reach forward with the lower jaw to nurse. The result is similar to that accomplished by Robin's orthostatic feeding program. One case was presented. The authors felt that the presence of the cleft palate was "no doubt responsible for alleviation of the marked dyspnoea so often present in these cases." The authors' theory regarding the embryology of this condition is quoted elsewhere.

La Page,[16] in 1937, presented one case of a 2-week-old infant with a receding chin, frequent attacks of cyanosis, difficulty breathing, and duskiness between attacks. This patient did not have a cleft palate. He was noted to do best lying on his face. The Eley-Farber splint was unsatisfactory. The patient died. This author recognized the advantage of positioning but felt that treatment must be directed toward "making the lower jaw develop"; otherwise he feared the likelihood of an adult problem. He recommended the Davis-Dunn lip guard and orthostatic feeding.

A unique method of skeletal traction was used in one case, as described by Callister[4] in 1937. This patient had a cleft palate. The Davis-Dunn appartus and the Eley-Farber brace were tried unsuccessfully. A thick silver wire was passed through the skin and around the mandible. This was in turn attached to a fixed flat ring brought around in front of the mouth, and as the

infant lay on his back countertraction was accomplished. The device was used for four weeks.

Another reference in the otolaryngology literature appeared in 1940. Schwartz [28] cited one patient, with cleft palate, who died of bronchopneumonia. Weisengreen and Sorsky [34] also presented a case in 1940. The condition of the palate is not mentioned. The patient died at the age of 52 hours after unsuccessful transfixion of the tongue. They felt that these cases require emergency treatment at birth, or therapeutic intervention later, when insufficient nutrition has inhibited normal development.

Llewellyn and Biggs [18] reported one case, in 1943, which had a cleft palate and which they treated by positioning, gavage feeding, and, later, a small brace which supported padded vertical bars which pressed the rami forward. They proposed as causes congenital syphilis, hereditary dystrophy from alcoholism, various infections, malnutrition of the embryo, and heredity. They subscribed to the Davis-Dunn theory of embryologic mechanism.

In 1946 Beverly Douglas [7] reviewed the literature, described what has become a standard procedure for the management of this condition, and presented six cases. In 1950 Douglas [8] presented six more cases, all of which were good results except one which died from "other causes." He also tabulated the results of a questionnaire sent to many of his colleagues. There were 21 nonoperative and 25 operative cases. There were 13 deaths in the former group and no deaths in the latter. Of the total 46 cases, 27 had cleft palate. The author stated: "Half the patients who died with conservative treatment were reported to have died suddenly before the nurse could get the pediatrician, and some were found dead by the attendant." He quoted Christie as follows:

The importance of including Micrognathia with abnormal motility of the tongue in our differential diagnosis whenever cyanosis, dysphagia, vomiting, or dyspnea is seen in a small infant cannot be overemphasized. Its correction by the means Dr. Douglas suggests in his operation has been life saving for several infants whom I have watched. The deformity is more common than I would have imagined until I became alert to its possibility.

Again in 1956 Douglas [9] reiterated the diagnostic points and treatment of micrognathia. He stated that in practically all of these babies rapid growth of the mandible occurs when malnutrition is corrected, by relief of the obstruction, by suturing the tongue to the lip. He cited the incidence of associated cleft palate in his series as 58% and in the Kiskadden-Dietrich series as 80%. Douglas reported 47 operative cases (by himself and others) with two deaths, giving a 4.5% mortality. The description of his operation is quoted from this paper (Fig. 7).

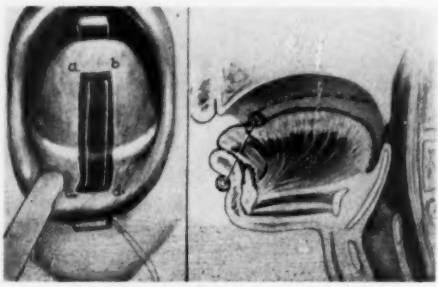


Fig. 7 (after Douglas: "The Treatment of Micrognathia with Obstruction by a Plastic Operation," Lyon chir. 52:420-431, 1956).—Illustration of technique of the Beverly Douglas operation.

With the baby under light ether anesthesia through a tube in the nose, the skin is prepped with weak iodine and alcohol, or other convenient media; zephiran, aqueous, or 1% silver nitrate is applied sparsely to the mucous membrane field. A silk suture is first placed through the tongue for traction at a distance of 0.5 cm. from the tip. It is held forward by this stitch in order to prevent retroposition during operation. After injection of saline immediately beneath the mucous membrane, a rectangular area of full-thickness mucous membrane extending along the undersurface of the tongue, the gum, and the posterior surface of the lower lip is outlined by a silk stitch at each corner, as follows: one stitch at each end of a transverse line, a-b, [Fig. 7] 1.5 cms. to 1.75 cms. in length on the underside of the tongue, one at each end of a similar transverse line, c-d, 1 to 1.5 cm. long, placed 1 cm. posterior to the free convex border of the lower lip. The mucous membrane of the underside of the tongue, extending down into the sulcus between the gum and lip and upon the posterior surface of the lip to the line cd is excised

in full thickness with a small scalpel in one rectangular piece by making traction on the stitches. It is important to avoid the orifices of Wharton's ducts in the sulcus which ducts may be identified and circumscribed, or as an alternative, the mucous membrane excision may be interrupted leaving a narrow transverse strip in the sulcus at their locations. The raw surface of the tongue is now brought down over the freshened gum into apposition with the raw surface of similar size on the posterior surface of the lower lip by sutures as follows: A lateral mattress tension suture of medium silkworm gut or "0" nylon enters the skin on the left side of the depression or sulcus of the lower lip approximately 1 cm. below the free border of the gum, just avoiding it. It then enters the raw surface of the underside of the tongue 1 mm. from the left-hand edge and about 2 mm. from the outer corner b, passes through the body of the tongue and out through the mucous membrane of the dorsum. A section of Carrel tubing of a size ordinarily used for Dakin's solution irrigations, or a soft rubber catheter of equal size (12 or 14 French) is cut about 1.5 cms. in length or slightly longer than the width between the ends of the mattress suture. The stitch is threaded through this tube and passes back through the tongue and lip on the right side in similar relation to these parts as before. The stitch is tied over the rubber tube in the normal depression of the lower lip not too tightly but with sufficient force to obtain good contact of all raw surfaces. A few 4-0 nylon or silk sutures on atraumatic needles are set on opposite edges of the lip and tongue before making traction on the mattress suture. These are finally tied so as to provide approximation of the mucous membrane edges and prevent entry of saliva and rolling in of edges. Recently, several surgeons have reported performing the operation very satisfactorily under local anesthesia. The sutures at the borders are left in about one week, and the mattress suture can usually be removed in 10 days.... After four or five months a simple "Z" type plastic operation is sufficient for complete release of the tongue-tie and any possible scarring of the lip may be easily remedied at this time. Actually, the release of the tongue may be accomplished at any time after a few weeks, but may be postponed until the lower teeth begin to erupt.

Sweet and Kemsley [31] reported two cases in 1947. Both cases had a cleft palate. They were treated by positioning and a padded wire splint which held the mandible forward and, also produced pressure sores at the angle of the mandible. A special nipple was devised to overcome the feeding difficulty. This consisted of a fine rubber catheter which projected ¾ in. beyond the end of the nipple. It was apparently used successfully in one case. The authors accepted the Davis and Dunn theory regarding the embryologic mechanism of development of this anomaly. They also agreed with the above authors, that the palatal cleft alleviated the dyspnea. One further statement, "The operation to anchor the tongue to the floor of the mouth always leads to bronchopneumonia," was quite remarkable at this date.

In 1948 May and Chun [20] reported a case of retruded mandible which was seen at the age of 5 days. This patient had a cleft palate. Sutures were placed through the tongue and attached to adhesive tape on the chest. The sutures cut through the tongue in two days. On the 9th day of age a tracheotomy was done and maintained for one month. At the age of 64 days, the day following a plastic procedure on the tongue, there was sudden respiratory obstruction, which was relieved by bronchoscopy, followed by another tracheotomy, which was maintained for 26 days. No further trouble was experienced.

Nisenson [22] presented one case of receding chin and glossoptosis associated with a cleft of the soft palate and uvula. This case was seen at the age of 7 weeks with a complaint of difficulty swallowing and breathing since birth, and cyanotic spells for one day. The case was managed satisfactorily with postural treatment, orthostatic feeding, and a headgear which purportedly held the rami forward. The child was able to be dismissed in 13 days and he wore the brace for one month. At the end of a year the author reported "still some recession of the mandible."

Longmire and Sanford [19] described a technique of traction, in 1949, by which they felt they could stimulate growth of the mandible. They stated: "Micrognathia if uncorrected may be extremely disfiguring in later life, and treatment of the deformity in infancy seems indicated whenever feasible." Their case was a baby with marked hy-

poplasia of the mandible and cleft palate, on whom the Beverly Douglas procedure had been performed at the age of 4 hours. At the age of 2 months a tantalum plate was inserted behind the symphysis through a small incision beneath the chin. A tantalum wire was attached and brought out through the mandible and the skin, passed over a pulley, and fastened to a 15 gm. weight. This device was maintained for three months, after which the authors reported that the length of the mandible seemed normal.

Sjölin [30] published an article in 1950 in which he reviewed the literature and reported two cases which were managed by positioning. One case had a cleft palate; the second apparently did not, judging by the author's implications.

In 1953 Kiskadden and Dietrich [15] reviewed the subject, with emphasis on the treatment of micrognathy. They credited Shukowsky with the first tongue-to-lip suturing procedure and felt that the present procedure should be termed the Shukowsky-Douglas operation. The authors concluded that the mechanical obstruction must be relieved, and they believed that with natural growth and weight gain there would be sufficient forward growth of the mandible to bring the tongue out of the hypopharynx and correct the functional anatomical deformity. They reviewed 25 cases. Five deaths occurred in the group of 15 cases treated by positioning alone. Failure to gain weight was considered one criterion of unsatisfactory management. They expected spontaneous correction of the receding chin at the age of 4 to 6 years, and in 16 patients whom they were able to trace this was the case.

Three cases were presented by Pruzansky and Richmond [24] in 1954, all with cleft palate. The first was tracheotomized, and the authors felt that, had the tracheotomy been done earlier, some of the several infections might have been avoided. The second case had a microglossia which helped avoid some of the potential respiratory problems. The authors concluded that use of the lip guard or traction on the symphysis does nothing to promote mandibular growth. They did feel that the important and prime objective is to provide an airway.

Beers and Pruzansky [1] presented one case in 1955 in which they studied the growth of the head. This child, with a cleft palate, had the Douglas operation performed at the age of 2 days, with dramatic results. The authors stated that "although tracheotomy has definite merit in the management of these infants it does require extended hospitalization and expert nursing care."

Champion [5] summarized the treatment of cleft palate associated with micrognathia, in 1956. He reported two cases in which palate repair was combined with the Beverly Douglas procedure. He felt that palate repair is the early surgical treatment of choice. He credited the Beverly Douglas procedure as being "very effective in a limited number of cases." Regarding tracheostomy, he decided that it is difficult to perform and only a temporary expedient, and summarized with "this line of treatment is undesirable."

Report of Cases

CASE 1.—Caucasian girl. Birth weight 7 lb. 11¾ oz.; micrognathia with cleft palate.

The patient was admitted to Children's Hospital of the East Bay, April 18, 1947, at the age of 6 weeks, weighing 6 lb. 10 oz. It was noted that "while on back breathing is difficult with sub- and suprasternal retractions." The plastic surgery consultant described moderate micrognathia with complete cleft of the hard and soft palate. He advised positioning, to be followed by the Beverly Douglas operation if results were not satisfactory. Dismissal was at the age of 11 weeks, weight 7 lb. 1 oz. At the age of 3½ years, on admission for staphylorraphy, the patient was still diagnosed as micrognathia, but no description of the mandible was given.

CASE 2.—Caucasian boy. Birth weight 7 lb. 4 oz.; micrognathia.

The patient was admitted to Children's Hospital of the East Bay, Nov. 14, 1947, at the age of 4 weeks, weighing 7 lb. 3 oz., after vomiting for one week. The consultant described a "small lower jaw," which he felt led to aerophagy. A lip-guard (Davis-Dunn) nursing bottle was suggested.

Fig. 8 (Case 3).—At the age of 6 months (left), and 9 years (right).

The patient was dismissed after one week weighing 7 lb. 3 oz. On several readmissions no further trouble was attributed to the micrognathia.

CASE 3.—Caucasian boy (Fig. 8).* Birth weight 3 lb. 1 oz.; micrognathia with cleft palate.

The patient was admitted to Children's Hospital of the East Bay, Nov. 28, 1948, at the age of 2 hours. A progress note on the third hospital day suggested "plastic and ear, nose and throat consultation when the patient is strong enough"! The next 37 days were marked by intermittent spells of cyanosis. The treatment consisted of positioning and gavage feeding. The patient was dismissed at the age of 40 days, weighing 5 lb. 6½ oz. The plastic surgery consultant had advised tongue suturing. The day following dismissal the patient was readmitted after he choked, turned blue, and was revived by the local fire department. A diagnosis of aspiration pneumonia was made. There were two more episodes of choking and cyanosis during the hospital stay, both relieved by laryngoscopy and aspiration. On the 41st hospital day the Beverly Douglas operation was performed. The patient was dismissed on the 15th postoperative day, at weight 7 lb. 6 oz. At the age of 2 years the following note appeared on a readmission record, "Mandible small, occlusion fair."

CASE 4.—Caucasian girl (Fig. 9).* Birth weight 8 lb. 10 oz.; micrognathia.

* Henry S. Patton, M.D., Oakland, Calif., furnished photographs of Cases 3 and 4.

The patient was admitted to Children's Hospital of the East Bay, June 30, 1949, at the age of 6 days, weighing 8 lb. 4 oz., because of inability to eat properly. The infant presented a "small mandibular bone and distinctive recessive chin," and there was moderate sternal retraction. She was dismissed nine days later, weighing 7 lb. 14 oz. The patient was readmitted at the age of 2 months with a diagnosis of malnutrition, weighing 8 lb. 9 oz. Six weeks later the Beverly Douglas procedure was performed, and the patient was dismissed on the 14th postoperative day at a weight of 10 lb. 12 oz. The tongue was released at the age of 13 months.

CASE 5.—Caucasian boy. Birth weight 6 lb. 11 oz.; micrognathia.

The patient was admitted to Children's Hospital of the East Bay, April 25, 1951, at the age of 5 months, weighing 14 lb. The complaint was "inability to eat well, gags on spoon feeding, always noisy breathing." The plastic surgery consultant noted the mandible to be 1.4 cm. posterior to the maxilla. He advised the use of the Davis-Dunn apparatus. On several readmissions, to the age of 3 years, no further mention was made of the micrognathia.

CASE 6.—Caucasian girl. Birth weight 7 lb. 5 oz.; micrognathia with cleft palate.

The patient was admitted to Children's Hospital of the East Bay, July 7, 1951, at the age of 19 days, weighing 7 lb. 8 oz. She was described as

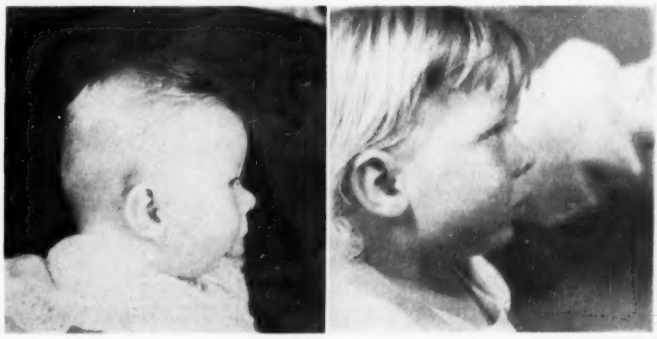


Fig. 9 (Case 4).—At the age of 6 months (left), and 7 years (right).

having "moderate micrognathia with rather marked falling back of the tongue in the supine position." The patient experienced intermittent respiratory obstruction, relieved by being placed on her face or side, and by care with her feeding. The Beverly Douglas operation was proposed in case the condition did not stabilize. Dismissal occurred after six days' hospitalization, at a weight of 7 lb. 13 oz. This child was readmitted at the age of 2 years and again at 5½ years, at which times no mention was made of micrognathia.

CASE 7.—Caucasian boy. Birth weight 5 lb. 13 oz.; micrognathia and cleft palate.

At the age of 5 weeks the weight was only 5 lb., and the patient was readmitted to the hospital in which he was born. He was admitted to Children's Hospital of the East Bay, Oct. 8, 1951, at the age of 12 weeks, weighing 7 lb. 8 oz., for performance of the Beverly Douglas operation. Postoperatively breathing was improved, but gavage feeding was required for 12 days. The tongue sutures tore out on the sixth postoperative day. At the age of 5 years this child is noticeably, but not unpleasantly, micrognathic.

CASE 8.—Caucasian boy. Birth weight 4 lb. 5 oz.; micrognathia.

The patient was admitted to Children's Hospital of the East Bay, Nov. 6, 1952, at the age of 4 days, weighing 3 lb. 13 oz., with the complaint of difficulty in feeding. Gavage feeding was used, and later the mother was taught to feed the baby carefully. The Davis-Dunn apparatus was found to be unsatisfactory in this case. The patient was dismissed on the 46th hospital day, weighing 5 lb. 8 oz. There was no mention of respiratory difficulty. The nurses' notes repeatedly mentioned "swallowing lots of air—lots of regurgitation."

CASE 9.—Caucasian boy. Birth weight 5 lb. 3½ oz.; micrognathia with cleft palate.

The patient was admitted to Children's Hospital of the East Bay, Nov. 15, 1955, at the age of 2 weeks, weighing 4 lb. 14½ oz., with a notation, "poor feeding because of cleft palate—transferred because of weight loss." The patient was fed by gavage and Breck feeder and dismissed in nine days weighing 5 lb. 3 oz. He was readmitted 18 days later, weighing 5 lb. 1½ oz., because of two spells of severe cyanosis during the previous four days, each requiring emergency hospital care. It was noted that his poor color and sternal retractions were improved by pulling the mandible forward. The ear, nose, and throat consultant advised positioning and careful feeding. In spite of this program the patient had repeated spells of apnea. On the fourth day of hospitalization, during a critical episode of apnea, one of the very capable pediatricians inserted a No. 00 Magill endotracheal tube through a laryngoscope and taped it in place. Three days later a tracheotomy was performed immediately after it was determined that traction on the tongue did not assure a safe airway. Five and a half months later the Beverly Douglas operation was performed and one month after that the tracheotomy tube was removed. Progress has been satisfactory. At the time of writing he is 21 months of age.

The technique of tracheotomy used in this and the next case is as follows: Nitrous-oxide-oxygen anesthesia is administered through an endotracheal tube. The anterior neck is prepared in the usual manner, with the head extended by placing a small towel roll under the shoulders. A level midway between the superior border of the sternum and the cricoid cartilage is selected and marked by the injection of 1% lidocaine (Xylocaine) with epinephrine (Adrenalin). A few cubic centimeters of the solution is infiltrated subcutaneously, and then deeply over the trachea. A 2 to 3 cm. horizontal incision is made across the midline through the fat and down to the deep fascia. The subcutaneous tissue is freed from the fascia and retracted. A 1½ cm. vertical incision is made through the fascia, in the midline, and blunt dissection is then carried out, directly down to the trachea, keeping at or slightly superior to the originally selected level. Skeletonization of the trachea is avoided. A vertical incision is then made through the pretracheal fascia and underlying two tracheal rings. This incision is separated with a mosquito forceps and the appropriate tracheotomy tube inserted (in these cases No. 0) after withdrawal of the endotracheal tube to the upper limits of the tracheal incision. A single silk suture may be taken at each end of the skin incision. The tracheotomy tube is secured by tying the tapes snugly with a square knot. The endotracheal tube is removed.

CASE 10.—Caucasian girl. Birth weight 5 lb. 4 oz.; micrognathia with cleft palate.

The patient was admitted to Children's Hospital of the East Bay, March 14, 1956, at the age of 11 days, weighing 5 lb. The patient was transferred from the maternity hospital because of "feeding problem—tongue would fall back in throat." The baby had been and was kept constantly in oxygen. The plastic surgeon advised tongue surgery, but the pediatrician preferred "conservative treatment." Three days later the patient had a sudden cyanotic episode. Four days thereafter the Beverly Douglas operation was performed (age 18 days, weight 5 lb. 3½ oz.). The patient did well until the second postoperative day, when the tongue sutures pulled out completely and the preoperative conditions recurred. During the ensuing five days there was a loss of a few ounces of weight, and the patient's color was described as less satisfactory. The plastic surgeon

advised tracheotomy. The pediatric consultant advised "a few days more." There was no weight gain, and three weeks later the patient had a sudden spell of apnea after gavage feeding. Heart sounds could not be heard. She was finally revived after administering intracardiac epinephrine. The following day during a discussion regarding the advisability of performing a tracheotomy the patient had another spell of apnea. The tracheotomy was performed immediately thereafter. Six weeks later the Beverly Douglas operation was repeated. The patient was extubated on the 18th postoperative day, and the tongue was released approximately 7½ months after surgery.

CASE 11.—Caucasian girl. Birth weight 6 lb. 14½ oz.; micrognathia with cleft palate.

The patient was admitted to Children's Hospital of the East Bay, Oct. 3, 1956, at the age of 12 hours with feeding and breathing difficulty. Initial plastic surgery consultation noted insufficient indication for tongue surgery; however, on the 10th day (weight 6 lb. 11 oz.) the Beverly Douglas operation was performed because of a deterioration of the patient's condition during the preceding 18 hours. Thereafter the patient steadily improved. The tongue sutures were removed on the ninth postoperative day. The patient was dismissed on the 24th postoperative day, weighing 7 lb. 12 oz.

CASE 12.—Caucasian boy. Birth weight 6 lb. 10 oz.; micrognathia.

The patient was admitted to Children's Hospital of the East Bay, Oct. 21, 1956, at the age of 5 days, weighing 5 lb. 8½ oz., with marked difficulty breathing. According to the plastic surgery consultant the obstruction was due to micrognathia and glossoptosis, and he advised the Beverly Douglas operation. On the same day (at the age of 1 month) laryngoscopy was performed by the ear, nose, and throat consultant, who described a "spastic cricopharyngeus" and suggested a central cause for the problem. The patient died at the age of 2 months after many spells of apnea and cyanosis and repeated diagnoses of pneumonitis. His final weight was 6 lb. 12 oz. He had multiple congenital anomalies, namely, arthrogryposis, micrognathia, and possibly hypoplasia of the pituitary gland. There was bilateral bronchopneumonia and there were focal superficial ulcers of the esophagus, attributed to a secondary Candida invasion; the effects, presumably, of prolonged antibiotic therapy.

CASE 13.—Caucasian boy. Birth weight 6 lb. 11 oz.; micrognathia.

The patient was admitted to Children's Hospital of the East Bay, May 6, 1957, at the age of 9 weeks, weighing 6 lb. 13 oz. The complaint was "difficulty swallowing because of mucus—has not gained weight." The condition was summarized on the eighth hospital day as "growth failure, feeding problem, congenital heart disease, pulmonary infection." The ear, nose, and throat consultant described respiratory obstruction due to micrognathia and glossoptosis and advised direct laryngoscopy with preparation to do a tracheotomy, as the obstruction was only partially relieved by pressing the rami forward. The plastic surgeon concurred in the diagnosis and initial management. The pediatric compartment was more impressed by the possible cardiac and evident pulmonary problems, and apparently could not accept the factor of mechanical obstruction as being significant. They chose to use positioning, gavage, and antibiotics. After repeated episodes of obstruction, aspiration, and cyanosis the patient expired at the age of 17 weeks, weighing 7 lb. 5½ oz. The pathologist reported congenital heart disease (interatrial, interventricular septal defects, and patent ductus), acute pulmonary edema, and bronchopneumonia.

There are 120 cases reported in the articles reviewed in this paper. My cases make a total of 133. There may be some duplication of cases in using the figures compiled by Douglas from correspondence with his colleagues. It is certain there are many cases unreported for reasons including early, undiagnosed, sudden death, on the one hand, and relatively mild symptoms, on the other. The range of cases presented, however, is quite extensive, from the mildest to the severest, and adequately serves to demonstrate the pertinent features of diagnosis and management (Table).†

Comment

Congenital micrognathia is a recognized syndrome, bearing the name of Pierre Robin who first described it, consisting of hypoplasia of the mandible and consequent glossoptosis, and resulting in hypopharyngeal obstruction. Breathing and feeding problems necessarily follow. Cleft palate is a finding in a majority of cases—68% of the cases reviewed in this paper. It would seem that the palatal cleft permits a greater degree of glossoptosis, whereas the presence of an intact palate serves to keep the tongue downward and a little more forward. Thus, Champion's method of early palate repair

† Since the completion of this paper two cases of congenital micrognathia were reported by Goff in the December, 1957, issue of The Annals of Otology, Rhinology and Laryngology. One case had an associated cleft palate. Both cases were tracheotomized, and in both cases the Beverly Douglas operation was performed.

Summary of Pertinent Factors from Case Histories

Author	No. of Cases	Cleft Palate	Treatment: Primarily Positioning	Treatment: Brace or Traction	Treatment: Douglas Operation	Treatment: Tracheotomy	Results: Survived	Results: Died
Lenstrup	3	3	3				3	
Janse	1	1		1		1		1
Eley and Farber	4	4	3	2		1*		1
Davis and Dunn	1	1	1				1	
La Page	1		1					1
Callister	1	1	1	1			1	
Schwartz	1	1	1					1
Weisengreen and Sorsky	1		1	1				1
Llewellyn and Biggs	1	1	1	1			1	
Douglas	68	40	21		47		55	13†
Sweet and Kemsley	2	2	2	2			2	
May and Chun	1	1		1	1	1	1	
Nisenson	1	1	1	1			1	
Longmire and Sanford	1	1		1	1		1	
Sjölin	2	1	2				2	
Kiskadden and Dietrich	25	20	21	6	4		20	5‡
Pruzansky and Richmond	3	3	2			1	3	
Beers and Pruzansky	1	1			1		1	
Champion	2	2			2§		2	
Weseman	13	7	7		6	2	11	2‖
Total	133	91	68	17	62	6	105	25

* Two hours after entry.
† Two of surgical group.
‡ None in surgical group.
§ Combined palate repair.
‖ None in surgical group.

might be expected to produce improvement in breathing as well as in feeding.

The most reasonable explanation, to date, of the embryologic mechanism of development of this condition is that of a nutritional defect occurring at a critical period of growth of the mandible. This transitory interruption of activity of its major growth center, if occurring before the end of the fourth month, could contribute to the failure of the palate to close. If the interruption occurs after the fourth fetal month, the palate will presumably have closed and resultant malpositioning of the tongue will have no effect upon it. This assumes that in this syndrome the palatal cleft is the result of a mandibular growth failure rather than a primary maxillary growth defect. Of speculative interest is the failure to find but one reference[8] to this syndrome in the Negro race.

The preponderance of opinion indicates that once the affected mandible resumes its growth, it continues to grow in a normal manner. Without knowing what the hereditary and environmental factors promise any individual child, it is impossible to state exactly how normal the recovery is. It seems reasonable to accept Brodie's opinion that the effects of an arrest are not compensated for by a subsequent acceleration of growth of the affected parts, but that, once recovery has occurred, growth resumes and continues in the usual manner. Thus the days of fetal repression are never made up, and there is always a relative deformity.

There is a notable reluctance of most authors to recommend or perform tracheotomy in these cases. This same reluctance is seen among the pediatric staff members and even otolaryngologists. They are confronted by a poor-appearing baby, with perhaps several congenital anomalies, and, presumably, a bleak future. The lack of enthusiasm for this poor-risk, poor-prognosis infant is understandable. Also, the pediatrician may feel that this infant isn't much worse than others who have survived,

as the evaluation of respiratory activity in this age group admittedly is difficult. He therefore is unwilling to permit surgical interference which promises, to him, new problems and prolonged specialized nursing care.

Unfortunately, neither attitude is realistic. If serious obstruction, constant or intermittent, exists, it must be recognized and relieved. The risk in performing a tracheotomy under proper conditions, available today, and with the help of antimicrobial and other refined supportive measures, is far less than the risk of nonsurgical management. The nursing care after tracheotomy becomes easier and more effective rather than more difficult. And, more important, the prognosis improves immediately.

Treatment should be directed primarily toward preserving life and promoting nutrition and growth. The use of various devices intended to stimulate growth of the mandible would seem to be as ill advised as the exclusive use of positioning and numerous cumbersome braces in an infant suffering repeated attacks of serious obstruction. There are instances where nothing short of immediate tracheotomy will save these patients. In these instances the Beverly Douglas operation can be expected to appreciably shorten the duration of the tracheotomy and consequent hospitalization. If the Beverly Douglas operation can be performed safely without tracheotomy, and if its results are satisfactory, it is the treatment of choice in cases in which positioning is not sufficient. There should be no hesitation in using tracheotomy before or after this procedure if it is necessary to insure a safe course. The risk of repeated attacks of aspiration and cyanosis is far greater than the risk of performing the Beverly Douglas operation or tracheotomy, or both.

Summary

Thirteen cases of congenital micrognathia are presented. Etiology and management are discussed. Positioning, the Beverly Douglas operation, and tracheotomy are stressed as appropriate therapeutic measures, singly or in combination, depending upon the severity of the symptoms.

2320 Channing Way (4).

REFERENCES

1. Beers, M. D., and Pruzansky, S.: The Growth of the Head of an Infant with Mandibular Micrognathia, Glossoptosis and Cleft Palate Following the Beverly Douglas Operation, Plast. & Reconstruct. Surg. 16:189-193 (Sept.) 1955.

2. Brodie, A. G.: On the Growth Pattern of the Human Head, Am. J. Anat. 68:209-262 (March) 1941.

3. Brodie, A. G.: Behavior of Normal and Abnormal Facial Growth Patterns, Am. J. Orthodontics 27:633-647 (Nov.) 1941.

4. Callister, A. C.: Hypoplasia of the Mandible (Micrognathy) with Cleft Palate, Am. J. Dis. Child. 53:1057-1059 (April) 1937.

5. Champion, R.: Treatment of Cleft Palate Associated with Micrognathia, Brit. J. Plast. Surg. 8:265-282 (Jan.) 1956.

6. Davis, A. D., and Dunn, R.: Micrognathia: Suggested Treatment for Correction, Am. J. Dis. Child. 45:799-806 (April) 1933.

7. Douglas, B.: The Treatment of Micrognathia Associated with Obstruction, Plast. & Reconstruct. Surg. 1:300-308 (Nov.) 1946.

8. Douglas, B.: A further Report on the Treatment of Micrognathia with Obstruction by a Plastic Procedure, Plast. & Reconstruct. Surg. 5:113-122 (Feb.) 1950.

9. Douglas, B.: The Treatment of Micrognathia with Obstruction by a Plastic Operation, Lyon chir. 52:420-431, 1956.

10. Eley, R. C., and Farber, S.: Hypoplasia of the Mandible (Micrognathy) as a Cause of Cyanotic Attacks in the Newly Born Infant, Am. J. Dis. Child. 39:1167-1175 (June) 1930.

11. Engel, M. B., and Brodie, A. G.: Condylar Growth and Mandibular Deformities, Surgery 22: 976-992 (Dec.) 1947.

12. Gladstone, R. J., and Wakeley, C. P. G.: Defective Development of the Mandibular Arch, J. Anat. 57:149-167 (Jan.) 1923.

13. Janse, M. H.: Macroglossia with an Associated Micrognathia Causing Respiratory Difficulty, Ann. Otol. Rhin. & Laryng. 39:99-101 (March) 1930.

14. Keibel, F., and Mall, F. P., Editors: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1910, Vol. 1, p. 86.

15. Kiskadden, W. S., and Dietrich, S. R.: Review of the Treatment of Micrognathia, Plast. & Reconstruct. Surg. 12:364-373 (Nov.) 1953.

16. La Page, C. P.: Micrognathia in the Newborn, Lancet 1:323-324 (Feb. 6) 1937.

17. Lenstrup, E.: Hypoplasia Mandibulae as Cause of Choking Fits in Infants, Acta paediat. 5:154-165, 1925.

18. Llewellyn, J. S., and Biggs, A. D.: Hypoplasia of the Mandible, Am. J. Dis. Child. 65:440-444 (March) 1943.

19. Longmire, W. P., Jr., and Sanford, M. C.: Stimulation of Mandibular Growth in Congenital Micrognathy by Traction, Am. J. Dis. Child. 78: 750-754 (Nov.) 1949.

20. May, H., and Chun, L. T.: Congenital Ankyloglossia (Tongue-Tie) Associated with Glossoptosis (Retruded Mandible) and Palatum Fissum (Cleft Palate), Pediatrics 2:685-687 (Dec.) 1948.

21. McCoy, J. D.: Factors Which Control the Treatment of the Dwarfted Mandible, Am. J. Orthodontics 25:850-864 (Sept.) 1939.

22. Nisenson, A.: Receding Chin and Glossoptosis, J. Pediat. 32:397-403 (April) 1948.

23. Orban, B.: Oral Histology and Embryology, Ed. 3, St. Louis, The C. V. Mosby Company, 1953, p. 13.

24. Pruzansky, S., and Richmond, J. B.: Growth of Mandible in Infants with Micrognathia, A. M. A. Am. J. Dis. Child. 88:29-42 (July) 1954.

25. Robin, P.: Backward Fall of the Root of the Tongue as Cause of Respiratory Disturbances, Bull. Acad. nat. méd. 89:37 (Jan. 2) 1923; abstracted, J. A. M. A. 80:805 (March 17) 1923.

26. Robin, P.: Glossoptosis Due to Atresia and Hypotrophy of the Mandible, Am. J. Dis. Child. 48:541-547 (Sept.) 1934.

27. Sarnat, B. G., and Gans, B. J.: Growth of Bones: Methods of Assessing and Clinical Importance, Plast. & Reconstruct. Surg. 9:140-160 (Feb.) 1952.

28. Schwartz, L.: Ateliosis of the Mandibular Arch, Arch. Otolaryng. 31:491-501 (March) 1940.

29. Scott, E. J.: An Experimental Study in Growth of the Mandible, Am. J. Orthodontics 24:925-934 (Oct.) 1938.

30. Sjölin, S.: Hypoplasia of the Mandible as a Cause of Respiratory Difficulties in the Infant, Acta paediat. 39:255-261, 1950.

31. Sweet, A. M., and Kemsley, M.: Micrognathia, Lancet 2:10-12 (July 5) 1947.

32. Warkany, J.: Congenital Malformations Induced by Maternal Nutritional Deficiency, J. Pediat. 25:476-480 (Dec.) 1944.

33. Warkany, J.: Congenital Malformations Induced in Rats by Maternal Nutritional Deficiency, Acta paediat. 36:223-229, 1948.

34. Weisengreen, H. H., and Sorsky, E. D.: Congenital Hypoplasia of the Mandible, J. Pediat. 16:482-486 (April) 1940.

Safe Anesthesia for Peroral Endoscopy

A Subsequent Report

WILLIAM P. KLEITSCH, M.D., Omaha

The agents most widely used to produce anesthesia for peroral endoscopy are tetracaine (Pontocaine) and cocaine. They are potent drugs and both are toxic in relatively small doses. When they are instilled into the tracheobronchial tree, the absorption of these agents approaches the speed of an intravenous injection. The combination of toxicity and rapid absorption makes untoward reactions to the anesthetic relatively common, and occasionally a death will result.[4]

Because the hazard has been recognized, such reactions to cocaine and tetracaine have been studied in detail.[1] The conclusions, which correspond to clinical experience, suggest the untoward reactions to these topical anesthetic agents are a result of overdosage rather than drug sensitivity. Rigid dose control is, therefore, required to reduce the hazards of toxicity. This is achieved by reducing the concentration of the solution and limiting the volume administered so that rigid maximum dose control is achieved.

Unfortunately, reducing the concentration of the solution also reduces its effectiveness. This results in a tendency to use more of the dilute solution, and thus dose control is lost. In attempting to arrive at a safe dose of either cocaine or tetracaine one finds himself faced with the problem of achieving effective concentration and adequate volume without exceeding the maximum safe dose. Carabelli attempted to overcome these problems with his micro-atomizer technique.[2]

The Central Office of the Veterans Administration has issued a dosage schedule for topically applied cocaine and tetracaine when used for peroral endoscopy.[5] It is their recommendation that an amount not to exceed 10 cc. be used of either tetracaine in a 1% solution or cocaine in a 10% solution. A preference was indicated for tetracaine, since the reactions are respiratory and nervous rather than cardiac as with cocaine.

Clinical experience with the recommended 1% tetracaine in 10 cc. doses proved it to be unsatisfactory in a high percentage of cases. Failures due to inability to abolish the cough reflex were common. As a result a search was made for a more effective and if possible a safer anesthetic agent for topical anesthesia.[3]

Having become aware of the use of tripelennamine (Pyribenzamine) as a topical anesthetic in cystoscopic procedures, we decided to study its potential as a topical anesthetic for peroral instrumentation.* This study seemed particularly appropriate since tripelennamine has a wide therapeutic range and overdosage could easily be controlled.

Accordingly several patients were prepared for bronchoscopy, using 10 cc. of a 2% tripelennamine solution applied to the pyriform sinuses and dropped into the tracheobronchial tree. It was discovered that this type of anesthesia was perfectly

Submitted for publication March 19, 1958.

Read before the 17th Congress of the International Society of Surgery, Mexico, D. F., Oct. 25, 1957.

From the Surgical Service of the Veterans Administration Hospital, Omaha, and of the Creighton University School of Medicine, and the University of Nebraska College of Medicine.

* The tripelennamine, Pyribenzamine, was made available by the Ciba Pharmaceutical Company.

adequate for bronchoscopy. There was one serious objection, however. Induction of anesthesia was relatively slow, and in this period of time most patients complained of substernal burning. In order to speed the induction of anesthesia without seriously adding to the toxicity of the solution 0.5% of tetracaine was added. The solution for topical anesthesia now consisted of 2% tripelennamine and 0.5% tetracaine. No more than 10 cc. of this solution was to be used for each patient. With this concentration of drugs it was found possible to produce topical anesthesia for all peroral endoscopic procedures with a rapid and uncomplicated induction. The suppression of the cough and gag reflex was universally effective.

Pleased by the effectiveness of this combination, we decided to test it further: First, all premedication was eliminated. This resulted in more cooperative patients and eliminated the postendoscopic depression which follows barbiturate and morphine narcosis. Secondly, the use of the anesthetizing solution was extended to the residents, so that the element of a skilled endoscopist was eliminated. Under these circumstances the tripelennamine-tetracaine solution continued to act as an effective topical anesthetic.

This solution has been employed for about four years with a great measure of success. Induction is rapid, and patient tolerance is good. Anesthesia is adequate for all peroral endoscopic procedures as well as punch biopsies, bronchograms, and the like. The amount of the mixture used is 10 cc., 5 cc. applied by means of sponges to the superior laryngeal nerves in the pyriform sinuses and 5 cc. instilled into the trachea. Thus a maximum dose of less than 200 mg. of tripelennamine and 50 mg. of tetracaine is used and only one-half of this dose is instilled into the tracheobronchial tree.

This anesthetic mixture has now been used in over 455 peroral endoscopic procedures. They are divided as follows:

Bronchoscopy	278
Bronchoscopy and bronchography	56
Esophagoscopy	33
Esophagoscopy and bronchoscopy	32
Laryngoscopy	32
Punch biopsies	5
Attempted endoscopy	19
Total	455

The attempted endoscopic procedures listed were those in which instrumentation was discontinued for one reason or another after anesthesia had been produced. The majority were repeated at a later date with a satisfactory outcome. Since most of the endoscopies in our hospital are done by residents, a number of different operators are involved. The anesthetic experiences are quite uniform in spite of the multiplicity of operators.

These experiences can be summarized as follows:

Depth of anesthesia—at least adequate. There is some opinion that it exceeds the anesthesia of cocaine.

Patient acceptance—a rare complaint of burning. No more than six such complaints in the entire series.

Ease of application—very satisfactory. The volume is sufficient to permit less meticulous technique than that required when cocaine or tetracaine alone are used.

Safety—apparently excellent. There was only one untoward incident in the series. A patient with a tumor in Rosenmüller's fossa after bilateral radical neck dissection and deep irradiation of the nasopharynx was prepared for rhinoscopy. While the nasopharynx was being sprayed, he developed a peculiar reaction which resembled a petit mal seizure. Electroencephalographic tracings, however, did not confirm this opinion. There was no evidence of a cause other than the local anesthesia. Nevertheless, a few days later the nose and nasopharynx were again prepared with the tripelennamine-tetracaine solution for rhinoscopy, which was carried out without incident. Whether the reaction was due to cerebral anoxia secondary to the straining or whether it was truly a drug reaction was never definitely established. If it were a true sensitivity to the drug, a second attempt should also have produced symptoms. That it may have been an overdose cannot be excluded except that large doses are not needed for nasopharynx anesthesia and in any event should not have reached the bronchial tree. Nevertheless, this episode is included as a reaction (untoward incident).

Comment

It seems to me that the important result of this study is to point out that anesthesia adequate for all peroral endoscopic procedures can be obtained by the use of antihistaminic drugs. It is unfortunate that the length of the induction period requires the addition of tetracaine to the tripelennamine solution. This, however, does not detract from the significance of the fact that the solution is an effective topical anesthetic. By itself 10 cc. of 0.5% tetracaine is totally incompetent to produce satisfactory topical anesthesia for peroral endoscopy.

This is not a so-called controlled series, but, as a matter of fact, it is controlled by past experience—experience which has taught us that overdosage with cocaine and tetracaine can be avoided only by exercising most rigid dose control, since the effective concentration of these drugs have such a narrow therapeutic range. This study suggests that perhaps a nontoxic topical anesthetic may yet be developed if the biochemical properties of the drugs in the antihistamine series are more completely evaluated.

Summary and Conclusion

A study is reported of the effectiveness of a solution containing 2% tripelennamine and 0.5% tetracaine as a topical anesthetic for peroral endoscopy in 455 instances. It was discovered that this solution is effective and apparently relatively safe as far as toxicity is concerned.

4101 Woolworth Ave. (5).

REFERENCES

1. Adriani, J., and Campbell, D.: Fatalities Following Topical Application of Local Anesthetics to Mucous Membranes, J. A. M. A. 162:1527-1530 (Dec. 22) 1956.

2. Carabelli, A. A.: Laryngo-Tracheo-Bronchial Anesthesia for Bronchoscopy and Bronchography with Pontocaine, Dis. Chest 15:532-545 (May) 1949.

3. Kleitsch, W. P.: A Safer Anesthetic for Peroral Endoscopy: Preliminary Report, A. M. A. Arch. Otolaryng. 59:195-197 (Feb.) 1954.

4. Palmer, E. D., and Deutsch, D. L.: Sudden Death During Preparation for Esophagoscopy with Tetracaine Gargle, Am. J. Digest. Dis. 22:95-96 (April) 1955.

5. Topical Anesthesia for Laryngoscopy, Bronchoscopy, Bronchography, Bronchial Aspirations and Gastroscopy, Veterans Admin. Tech. Bull. 10A-106, Jan. 16, 1948.

Neurinoma (Schwannoma) of the Facial Nerve

Report of Two Cases

JACK W. POU, M.D., Shreveport, La.

Neurinomas of the facial nerve are rare tumors. To my knowledge, only 20 cases have been recorded in the medical literature. Because of the rarity of this type of tumor, two additional cases will be reported. In one, the clinical course is believed to be unique. The other is thought to be the first case to be reported of neurinoma of the facial nerve in a patient who also had a neurinoma of the eighth cranial nerve.

Report of Cases

CASE 1.—A white woman 59 years old was seen on Dec. 24, 1955, because of severe constant pain, deafness, and drainage from the left ear. She was aware of a throbbing sensation in the ear which appeared to be synchronous with her heart beat. The pain had been present for approximately two years but during the past year had become progressively severer and on admission it was almost unbearable.

About four years before, the patient consulted a physician because of a feeling of fullness in the left ear. She was told that she had a polyp in her ear which had probably been there for many years. Two years later she started having some drainage from this ear and also a dull, constant, aching pain.

On examination on Dec. 24, a smooth, glistening tumor could be seen completely filling the auditory canal and protruding from the external auditory meatus (Fig. 1). There was an associated purulent discharge. The tumor was extremely sensitive to manipulation, and it bled slightly when manipulated. Audiometric study showed a 45 db. average hearing loss. The pneumatic mastoid process appeared normal in the roentgenogram of the mastoids. In view of this and the presence of a tumor in the auditory canal, biopsy was not performed preoperatively because a glomus jugulare tumor was considered a definite possibility. There were no clinical manifestations of involvement of the facial nerve.

On Jan. 18, 1956, an endaural incision was made, and the mastoid cortex was removed. The wall of the membranous canal was cut anteriorly so that

Submitted for publication April 30, 1958.

Presented as a candidate's thesis in partial fulfillment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Oct. 1, 1957.

Fig. 1 (Case 1).—Neurinoma of facial nerve completely occluding external auditory meatus.

Fig. 2 (Case 1).—Complete facial paralysis two months postoperatively.

Fig. 3 (Case 1).—Note 60% to 70% return of facial function 13 months postoperatively.

it could be folded back into the mastoid bowl for better exposure of the growth. The tumor had grown into the auditory canal through the wall of the posterior canal and had eroded through the tympanic membrane from the external canal into the middle ear. This portion of the tumor was elevated out of the middle ear; this left a clean posterior central perforation of the tympanic membrane. The middle ear and hypotympanum were then perfectly clean. The remaining wall of the posterior canal was taken down until the facial nerve was exposed. The tumor was carefully dissected off the nerve which arose from a wide base on the descending portion of the facial nerve.

When the patient first reacted after the operation, she had slight facial weakness but still could close her eye. The next morning complete facial paralysis was present. Within two months the mastoid cavity had completely epithelized; the perforation in the tympanic membrane had closed; the patient was free of pain and drainage, and her hearing was up to the 15 db. level. The facial paralysis was still present (Fig. 2). Nerve graft was advised, but the patient refused to submit to further surgical treatment since she had been completely relieved of pain. At this writing there has been approximately 60% return of facial function (Fig. 3). Incidentally, after the operation it was learned that the patient had had a small neurofibroma removed from the sole of her foot 12 years previously.

Comment.—To my knowledge, this is the first case of a facial nerve neurinoma of the auditory canal to be reported in which there was no preexisting facial paralysis. It demonstrates that even fairly far-advanced cases of facial nerve neurinomas do not necessarily produce facial paralysis. The severe pain of approximately two years' duration is also unusual. A preoperative diagnosis of facial nerve neurinoma was not made because biopsy was considered contraindicated in view of the possibility of a glomus jugulare tumor. The throbbing sensation noted by the patient was a pulsating discharge due to secondary infection of the middle ear.

CASE 2.—A white woman 28 years old was seen in July, 1954, complaining of a large mass bulging out of her left auditory canal. She had first noted the mass four years previously, and about the same time some swelling developed under the left ear in the region of the parotid gland. A biopsy was performed of the swelling in the neck, and the report was schwannoma. There was a constant draining fistula below the ear from the site of the biopsy. Since then the tumor in her ear had continued to increase in size and the fistula in her neck had continued to drain.

In 1948 an acoustic neurinoma on the left had been removed. The facial nerve was damaged during the operation and this resulted in complete facial paralysis on the side operated on.

On examination on admission complete facial paralysis on the left side, a draining fistula in the left side of the neck below the ear, and complete occlusion of the left auditory canal by a cystic fluctuant mass, which gave the appearance of arising from the floor or wall of the posterior canal, were noted. Audiometric study showed almost complete bilateral nerve deafness, with only slight hearing in the 250 and 500 frequencies. In the roentgenogram of the mastoid processes, the one on the left appeared to be completely destroyed.

In August, 1954, a radical mastoidectomy was performed in order to remove the growth. A large tumor was seen bulging up from the floor of the external canal and extending into the middle ear. The ossicles were completely eroded. The tumor apparently arose from the facial nerve in the descending portion. However, it was so extensive that it was difficult to determine the exact portion of the facial nerve from which it originated. The mass was dissected from above down and was found to have eroded the bony plate of the mastoid process and extended into the neck in the region of the parotid gland. There was a dehiscence of bone in the region of the jugular bulb. A postauricular incision was made and joined with the fistula that was already present in the neck. As much tumor as possible was removed from this region by means of blunt dissection. In the removal of neoplastic tissue from the region of the jugular bulb the vessel was entered; this caused considerable bleeding, which was controlled by packing. The facial nerve throughout the extent of the middle ear was completely eroded up to 1 cm. of the exit of the nerve from the cranium. The horizontal semicircular canal had been completely eroded; this caused destruction of the labyrinth.

The pathologist reported the tumor to be a schwannoma or neurinoma of the facial nerve. He reviewed the slides of the tissue removed in 1948, and reported that that tumor was the same type as the one removed from the facial nerve.

The postoperative course was uneventful. The fistula in the neck was closed and ceased to drain. The mastoid cavity completely epithelized, and at this writing the patient's ear is well healed.

Comment.—This is believed to be the first case of neurinoma of the facial nerve in a patient who also had a neurinoma of the eighth cranial nerve. The patient had facial parlysis due to an intracranial operation for an acoustic neurinoma, but she would have had facial paralysis because the facial nerve in the middle ear had been completely destroyed. Schröder [1] reported a case so extensive that it had eroded the bony plate of the tegmen and caused intracranial complications. This is the only case I could find in which the bony plate of the mastoid had been completely eroded and the tumor extended into the neck. This case also shows that recurrence of neurinomas is uncommon, for I am certain that all of this tumor was not removed, and after two and one-half years this patient has no signs of recurrence.

Neurinoma in General

Neurinoma, or schwannoma, is a benign tumor arising from the cells of Schwann of the neurilemma covering the nerve. It may develop in any nerve possessing a neurilemma. This includes cranial as well as sympathetic nerves. In Stout's [2] report of 50 cases, the largest collected series of neurinomas in the literature, only 26% were located above the clavicle. Most neurinomas are found in the neck rather than the head, owing to the relatively large number of peripheral nerve trunks grouped in the region of the neck.

The usual sites of formation of tumors in the cranial nerves are the unmixed sensory nerves, but tumors may arise also in the mixed nerves, usually on the sensory root. Since neurinomas rarely develop on cranial nerves containing only motor fibers, the relatively frequent occurrence of these lesions on the facial nerve is striking. Of the cranial nerves the auditory is the most frequently involved, and, according to Dandy,[3] "Cerebellopontine tumors are perhaps the most common of the benign encapsulated tumors of the brain." Cushing [4] stated that acoustic neurinomas represent from 8% to 9% of all intracranial tumors.

The cause of neurinomas is not known. However, Tronconi [5] believed that they are due to abnormal stimulation, or to unbalance of the neuritic-schwannian-fibroblastic system. They may develop at any time during life and they are encountered in both

sexes with equal frequency. The age distribution of cranial neurinomas is fairly definite. Craig, Keith, and Kernohan [6] found none in a review of 427 brain tumors of children. Bennett [7] found only 15 (3.4%) in a series of 446 intracranial tumors in persons between 18 and 38 years of age. According to Kernohan and Sayre,[8] they occur in persons between 36 and 50 years of age.

Review of Literature on Neurinoma in General

All tumors of nerves were classed as neuromas until 1863, when Virchow [9] differentiated between true and false neuromas. He pointed out that the false neuroma consists chiefly of connective tissue and he classified these as neurofibromas. According to Penfield,[10] Thompson subdivided these neurofibromas into two types: multiple false neuromas, such as are found in von Recklinghausen's disease, and solitary false neuromas.

The controversy that has raged through the years concerning the nature of the origin of the cells of tumors arising from nerve roots is evidenced in the varied and complicated nomenclature. The most widely used term in the literature is neurinoma, introduced by Verocay, according to Kettel.[11] Another popular term is schwannoma; its popularity is apparently due to the fact that most of the available evidence favors the schwannian theory of histogenesis. Tarlov,[12] Penfield,[13] and others, who employed various staining techniques, thought that these tumors were fibroblastic, arising from the connective tissue of the nerve bundles. Other terms that have been used to describe the same condition are perineural fibroblastoma,[14] neurilemmoma,[15] peripheral glioma, lemmoma, encapsulated neurofibroma, neurinoma sarcomatodes, false neuroma, and fibroma molluscum.

Review of Literature on Neurinoma of Facial Nerve

Roberts [16] ably reviewed the literature on neurinoma of the facial nerve in 1943. He was able to find reports of 12 cases up to 1940. Since all but one of these cases were reported in foreign periodicals, the information regarding these cases was taken from Robert's article.

According to Roberts,[16] the first case of neurinoma of the facial nerve to be reported in the literature is that of Schmid in 1930. Schmid's patient, a girl 16 years old, had a small tumor that could be seen in the auditory canal arising from the wall of the posterior canal. The tympanic membrane and hearing were normal, but facial paralysis was present. At mastoidectomy a tumor of the facial nerve in its descending portion was found.

The next year Schröder reported a case of neurinoma causing chronic facial paralysis and otitis media in a man 25 years old. A tumor completely filling the middle ear had destroyed part of the posterior auditory canal and mastoid process; it arose from the descending portion of the facial nerve.

Grossmann and Leidler reported another case in 1933. The patient had been seen in 1926 with right facial paralysis and a polyp in the right external auditory canal which arose from the wall of the posterior canal. The biopsy report of the polyp was leiomyoma. Treatment with radium caused some reduction in size of the tumor, but the facial paralysis persisted. Six years later the patient returned because of deafness, headache, and persistent facial paralysis. Destruction of the posterior bony canal with a normal pneumatized mastoid process was demonstrated roentgenographically. At operation a large tumor was seen originating from the descending portion of the facial nerve. The pathologic report was neurinoma.

The report of three additional cases in 1935 by Altmann brought the total number of reported cases of neurinoma of the facial nerve to six. Altmann's first case was a woman 26 years old with facial paralysis, conductive deafness, polypoid tumor in the auditory canal, and roentgenographic dem-

onstration of normal mastoid. The biopsy report was neurinoma. Irradiation caused no change in the physical findings or symptoms. The other two patients had permanent facial paralysis. Because of involvement of the middle ear and mastoid in one and a permanent dead labyrinth in the other, both were treated surgically.

Greifenstein's case, reported in 1936, is believed to be the first recorded case of facial nerve neurinoma arising from the horizontal portion of the facial nerve, as well as the first of a patient who did not have preexisting facial paralysis. It was discovered at postmortem examination. The patient's only symptom was slight conductive hearing loss. Greifenstein concluded that symptoms appear only after the neurinoma has reached a considerable size.

In 1937 Ohnishi described a neurinoma that arose from the descending portion of the facial nerve and presented the usual symptoms of facial paralysis and conductive deafness. An additional case reported in the same year by Altmann brought the total number of reported cases to nine.

The first case of neurinoma of the facial nerve to appear in the American medical literature is that of Williams and Pastore [17] in 1939. It is also believed to be the first recorded case of neurinoma originating from the horizontal portion of the facial nerve causing symptoms. The tumor had destroyed the labyrinth and invaded the mastoid cells. Williams and Pastore pointed out that if a specimen is not obtainable from the aural canal for biopsy, in the presence of a destructive process involving the mastoid bone, a diagnosis of a malignant process cannot be ruled out without performance of an exploratory operation.

In 1939 Rejtö also described a neurinoma of the horizontal portion of the facial nerve; in this case the labyrinth was not involved.

The next year Vysotskaya reported a case of neurinoma arising from the descending portion of the facial nerve and producing the usual symptoms of facial paralysis and conductive deafness with a polypoid tumor in the auditory canal. The same year Kos reported a case of neurinoma involving the descending portion of the facial nerve discovered at autopsy.

The second case of neurinoma of the facial nerve causing symptoms to appear in the American medical literature is that of Roberts.[16] The patient was a white man 50 years old with a discharging ear for 15 years, deafness for 20 years, and paralysis of the right side of the face for 30 years. At operation an extensive neurinoma arising from the horizontal portion of the facial nerve was discovered.

In 1944 Bogdasarian [18] described another case of neurinoma arising from the horizontal portion of the facial nerve. The drum was bulging, and after myringotomy the tumor prolapsed into the auditory meatus. The diagnosis was made preoperatively by biopsy. Bogdasarian pointed out the fact that tumors originating from the horizontal portion of the facial nerve are more likely to cause conductive deafness before facial paralysis because there is more room for expansion into the middle ear. Since there is less room for expansion in the descending portion of the nerve, the facial paralysis develops earlier. The following year Skoog [19] reported a case of neurinoma of the descending portion of the facial nerve.

In 1946 Kettel [11] reported what is believed to be the first case of neurinoma causing pain. This was associated with facial paralysis, which disappeared only to recur and persist. The tumor was not visible in the middle ear or auditory canal, and hearing was normal. Operation revealed a neurinoma of the descending portion of the facial nerve.

In 1947 Lundgren [20] reported one case and Loeliger [21] reported two cases of neurinoma arising from the descending portion of the facial nerve and producing the usual symptoms of facial paralysis, conductive deafness, and draining ears.

The most recent case to be reported is that of Rainer,[22] who described a neurinoma

arising from the horizontal portion of the facial nerve and spreading to the tympanic cavity, causing deafness, twitching, and paralysis. He emphasized that, if operation can be done early, paralysis can be prevented.

Comment.—A review of the literature on neurinoma of the facial nerve indicates that the clinical picture usually follows a fairly definite pattern, with a few variations. The tumor usually occurs in young to middle-aged persons. The first, and frequently for a long time the only, symptom is facial paralysis, which may develop gradually or occur suddenly, and is usually permanent. A tumor eventually can be seen in the external auditory canal, appearing to arise from the wall of the posterior canal. The facial paralysis is usually present prior to the discovery of the tumor. The tympanic membrane and hearing remain intact for some time. The tumor grows expansively, and impairment of hearing may be due to mechanical obstruction or to otitis media resulting from ingrowth of the tumor into the middle ear. The tumor usually causes no pain. If it arises from the horizontal portion of the facial nerve, conductive deafness may occur prior to facial paralysis. Only five cases have been reported originating from the horizontal portion of the facial nerve. The site of origin is usually in the descending portion.

The lesions found at operation have ranged from a minor, well-defined focus around the pars descendens [11,22] to extensive destruction of the mastoid process inclusive of the posterior and even the anterior wall of the auditory meatus, the middle ear, the labyrinth, and the pyramid,[23] and in one case the tumor had penetrated the internal ear, causing an abscess of the brain.[1]

Pathology

Gross Description.—Neurilemmomas vary considerably in size. They are usually encapsulated, fusiform, and smooth, although the surface may be nodular and even distinctly lobulated. The surface is usually gray, but small cysts may be seen shining through the capsule. On section the cut surface is gray and mottled and there are varying-sized yellow, orange, or brown areas. The majority contain smooth-walled cysts of varying sizes and number. Occasionally, on cross section, these tumors have the appearance of a sponge with little actual neoplastic tissue remaining. Calcium deposits are rare.

The *microscopic description* of neurinomas by Ehrlich and Martin [24] in 1943 has not been improved upon. They described four characteristic histologic features of neurilemmomas: (1) palisading of the nuclei (Fig. 4); (2) interlacing bundles of fibers (Fig. 4); (3) small pigmented regions and foci of degeneration, and (4) foam cells. The palisading of the nuclei is the most characteristic but least constant of the findings. The elongated delicate nuclei lie side by side in rows like a picket fence and between the rows of nuclei the fibrils of the cells are also parallel. The most constant but least significant finding is the interlacing of parallel bundles of cells and fibrils. This fibrillar polar arrangement of cells is usually referred to as the Antoni Type A tissue. The Antoni Type B tissue has a loose, reticular, apolar arrangement and represents the background in most of these tumors. The lipoid-containing foam cells are practically always present. This is thought to be degenerated neoplastic cells.

Differential Diagnosis

In the differential diagnosis must be considered chronic otitis media, chronic mastoiditis, carcinoma, tuberculosis, glomus jugulare, and early Bell's palsy. In chronic otitis media and chronic mastoiditis there is usually a history of chronic suppuration before facial paralysis occurs. In the early stages of development paralysis is usually the first and only symptom of neurinoma. Roentgenograms of the mastoid in the former diseases would show a sclerotic process, whereas in a neurinoma the mastoid cells often are pneumatic unless secondary in-

onstration of normal mastoid. The biopsy report was neurinoma. Irradiation caused no change in the physical findings or symptoms. The other two patients had permanent facial paralysis. Because of involvement of the middle ear and mastoid in one and a permanent dead labyrinth in the other, both were treated surgically.

Greifenstein's case, reported in 1936, is believed to be the first recorded case of facial nerve neurinoma arising from the horizontal portion of the facial nerve, as well as the first of a patient who did not have preexisting facial paralysis. It was discovered at postmortem examination. The patient's only symptom was slight conductive hearing loss. Greifenstein concluded that symptoms appear only after the neurinoma has reached a considerable size.

In 1937 Ohnishi described a neurinoma that arose from the descending portion of the facial nerve and presented the usual symptoms of facial paralysis and conductive deafness. An additional case reported in the same year by Altmann brought the total number of reported cases to nine.

The first case of neurinoma of the facial nerve to appear in the American medical literature is that of Williams and Pastore [17] in 1939. It is also believed to be the first recorded case of neurinoma originating from the horizontal portion of the facial nerve causing symptoms. The tumor had destroyed the labyrinth and invaded the mastoid cells. Williams and Pastore pointed out that if a specimen is not obtainable from the aural canal for biopsy, in the presence of a destructive process involving the mastoid bone, a diagnosis of a malignant process cannot be ruled out without performance of an exploratory operation.

In 1939 Rejtö also described a neurinoma of the horizontal portion of the facial nerve; in this case the labyrinth was not involved.

The next year Vysotskaya reported a case of neurinoma arising from the descending portion of the facial nerve and producing the usual symptoms of facial paralysis and conductive deafness with a polypoid tumor in the auditory canal. The same year Kos reported a case of neurinoma involving the descending portion of the facial nerve discovered at autopsy.

The second case of neurinoma of the facial nerve causing symptoms to appear in the American medical literature is that of Roberts.[16] The patient was a white man 50 years old with a discharging ear for 15 years, deafness for 20 years, and paralysis of the right side of the face for 30 years. At operation an extensive neurinoma arising from the horizontal portion of the facial nerve was discovered.

In 1944 Bogdasarian [18] described another case of neurinoma arising from the horizontal portion of the facial nerve. The drum was bulging, and after myringotomy the tumor prolapsed into the auditory meatus. The diagnosis was made preoperatively by biopsy. Bogdasarian pointed out the fact that tumors originating from the horizontal portion of the facial nerve are more likely to cause conductive deafness before facial paralysis because there is more room for expansion into the middle ear. Since there is less room for expansion in the descending portion of the nerve, the facial paralysis develops earlier. The following year Skoog [19] reported a case of neurinoma of the descending portion of the facial nerve.

In 1946 Kettel [11] reported what is believed to be the first case of neurinoma causing pain. This was associated with facial paralysis, which disappeared only to recur and persist. The tumor was not visible in the middle ear or auditory canal, and hearing was normal. Operation revealed a neurinoma of the descending portion of the facial nerve.

In 1947 Lundgren [20] reported one case and Loeliger [21] reported two cases of neurinoma arising from the descending portion of the facial nerve and producing the usual symptoms of facial paralysis, conductive deafness, and draining ears.

The most recent case to be reported is that of Rainer,[22] who described a neurinoma

arising from the horizontal portion of the facial nerve and spreading to the tympanic cavity, causing deafness, twitching, and paralysis. He emphasized that, if operation can be done early, paralysis can be prevented.

Comment.—A review of the literature on neurinoma of the facial nerve indicates that the clinical picture usually follows a fairly definite pattern, with a few variations. The tumor usually occurs in young to middle-aged persons. The first, and frequently for a long time the only, symptom is facial paralysis, which may develop gradually or occur suddenly, and is usually permanent. A tumor eventually can be seen in the external auditory canal, appearing to arise from the wall of the posterior canal. The facial paralysis is usually present prior to the discovery of the tumor. The tympanic membrane and hearing remain intact for some time. The tumor grows expansively, and impairment of hearing may be due to mechanical obstruction or to otitis media resulting from ingrowth of the tumor into the middle ear. The tumor usually causes no pain. If it arises from the horizontal portion of the facial nerve, conductive deafness may occur prior to facial paralysis. Only five cases have been reported originating from the horizontal portion of the facial nerve. The site of origin is usually in the descending portion.

The lesions found at operation have ranged from a minor, well-defined focus around the pars descendens [11,22] to extensive destruction of the mastoid process inclusive of the posterior and even the anterior wall of the auditory meatus, the middle ear, the labyrinth, and the pyramid,[23] and in one case the tumor had penetrated the internal ear, causing an abscess of the brain.[1]

Pathology

Gross Description.—Neurilemmomas vary considerably in size. They are usually encapsulated, fusiform, and smooth, although the surface may be nodular and even distinctly lobulated. The surface is usually gray, but small cysts may be seen shining through the capsule. On section the cut surface is gray and mottled and there are varying-sized yellow, orange, or brown areas. The majority contain smooth-walled cysts of varying sizes and number. Occasionally, on cross section, these tumors have the appearance of a sponge with little actual neoplastic tissue remaining. Calcium deposits are rare.

The *microscopic description* of neurinomas by Ehrlich and Martin [24] in 1943 has not been improved upon. They described four characteristic histologic features of neurilemmomas: (1) palisading of the nuclei (Fig. 4); (2) interlacing bundles of fibers (Fig. 4); (3) small pigmented regions and foci of degeneration, and (4) foam cells. The palisading of the nuclei is the most characteristic but least constant of the findings. The elongated delicate nuclei lie side by side in rows like a picket fence and between the rows of nuclei the fibrils of the cells are also parallel. The most constant but least significant finding is the interlacing of parallel bundles of cells and fibrils. This fibrillar polar arrangement of cells is usually referred to as the Antoni Type A tissue. The Antoni Type B tissue has a loose, reticular, apolar arrangement and represents the background in most of these tumors. The lipoid-containing foam cells are practically always present. This is thought to be degenerated neoplastic cells.

Differential Diagnosis

In the differential diagnosis must be considered chronic otitis media, chronic mastoiditis, carcinoma, tuberculosis, glomus jugulare, and early Bell's palsy. In chronic otitis media and chronic mastoiditis there is usually a history of chronic suppuration before facial paralysis occurs. In the early stages of development paralysis is usually the first and only symptom of neurinoma. Roentgenograms of the mastoid in the former diseases would show a sclerotic process, whereas in a neurinoma the mastoid cells often are pneumatic unless secondary in-

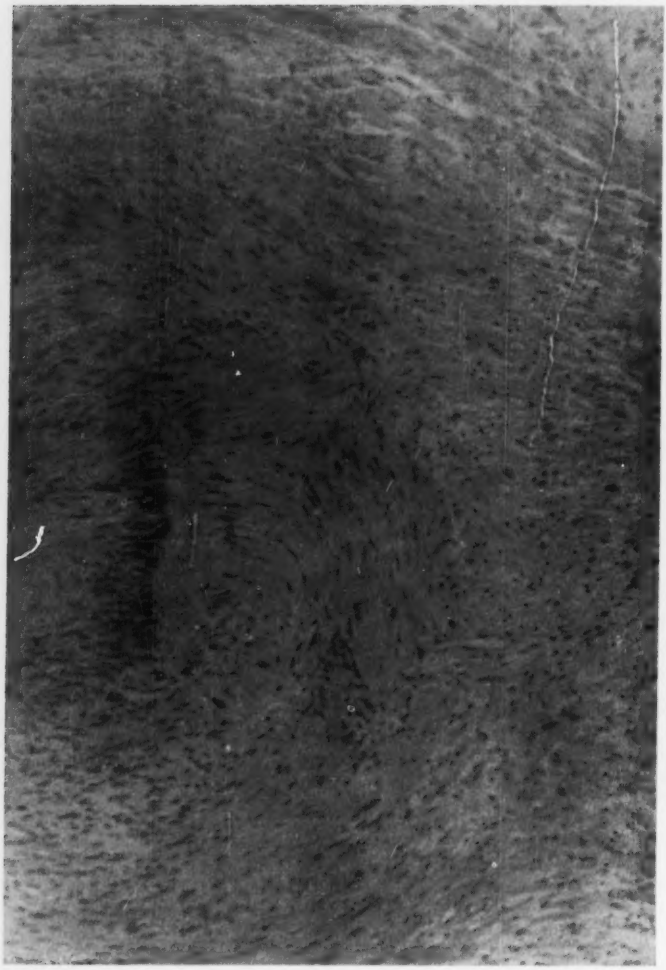


Fig. 4. — Photomicrograph of a neurinoma of the facial nerve. Note palisading of the nuclei and interlacing bundles of fibers.

fection has already occurred. Roentgenographic evidence of destruction of the posterior and inferior walls of the bony auditory meatus, the floor of the tympanum, and the lateral wall of the atticus is suggestive of tumor rather than chronic otitis media. The absence of other tuberculous changes makes the diagnosis of tuberculosis less probable. Carcinoma of the ear practically always develops as a result of chronic otitis media so that facial paralysis would occur late in the course of the disease, in contrast to early appearance of paralysis in neurinomas.

Glomus jugulare tumors may present a differential problem since they may be seen in the auditory canal and a normal pneumatic mastoid process may be visualized in the roentgenograms. As a rule, neurogenic tumors in the auditory canal cause preexisting facial paralysis. However, in one of the cases herein presented the patient had a tumor in the auditory canal with normal mastoid demonstrated in the roentgenograms but no preexisting paralysis. History of profuse bleeding ordinarily occurs with a glomus jugulare tumor.

It is extremely difficult to differentiate between a neurinoma that has not yet become visible in the auditory meatus and Bell's palsy. Bell's palsy is usually associated with pain in the beginning, whereas neurinoma is not. If facial function does not show signs of returning in six weeks to two months, then exploration may be the only means of making a differential diagnosis between Bell's palsy and neurinoma.

Treatment

Treatment of neurinomas is surgical removal. Irradiation has not been successful. There have been no reports of recurrence after surgical removal. Often during removal of the tumor, the continuity of the nerve will be broken over some distance. In such cases, at the time of removal it is desirable to do nerve grafting according to the method of Ballance and Duel.[25]

Prognosis

Neurinomas are benign tumors that are usually not a threat to life, although complications may arise late in the course of the disease, such as involvement of the labyrinth and brain, if the tumor is not removed. The prognosis as to future function of the facial nerve is poor.

Summary

Neurinoma of the facial nerve is a definite though rare clinical entity. Only 20 cases have been found in the literature.

Neurinomas are benign tumors producing symptoms by growth. The usual clinical manifestations are facial paralysis, followed by appearance of a tumor in the external auditory canal through the wall of the posterior canal. Conductive deafness may be the first symptom if the tumor arises from the horizontal portion of the nerve. Only five reports of cases of neurinomas originating in the horizontal portion of the facial nerve have been found in the literature.

The clinical picture of facial nerve neurinomas is sufficiently varied to warrant considering this type of tumor in the differential diagnosis of any tumor in the auditory canal. It should also be considered a definite possibility in cases of facial paralysis diagnosed as Bell's palsy in which function does not return within a reasonable length of time.

Treatment is surgical removal. The prognosis as to life is good. If the neurinoma is not removed, eventually the labyrinth and brain may become involved. Return of function of the facial nerve is not likely.

Two additional cases of neurinoma of the facial nerve have been reported. One is believed to be the first case reported in which the tumor presented into the external auditory canal without the patient having preexisting facial paralysis. The other is thought to be the first case of neurinoma of the facial nerve in a patient who also had a neurinoma of the eighth cranial nerve.

1513 Line Ave.

REFERENCES

1. Schröder, R., cited by Kettel.[11]

2. Stout, A. P.: Tumors of the Peripheral Nervous System, in Atlas of Tumor Pathology, U. S. Armed Forces Institute of Pathology, 1949.

3. Dandy, W.: Cerebellopontine (Acoustic) Tumors, in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1932, Vol. 12, Chap. 1, p. 517.

4. Cushing, H. W.: Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontine Angle, Philadelphia, W. B. Saunders Company, 1917, p. 17.

5. Tronconi, V.: On the Histological Structure of Some Neurinomas, or Schwannomas, Sc. med. ital. 1:507-512 (July-Sept.) 1950.

6. Craig, W. M.; Keith, H. M., and Kernohan, J. W.: Tumors of the Brain Occurring in Childhood, Acta psychiat. et neurol. 24:375-390, 1949.

7. Bennett, W. A.: Primary Intracranial Neoplasms in Military Age Group—World War II, Mil. Surgeon 99:594-652 (Nov.) 1946.

8. Kernohan, J. W., and Sayre, G. P.: Tumors of the Central Nervous System, in Atlas of Tumor Pathology, U. S. Armed Forces Institute of Pathology, Fascicle 35, 1952, pp. 76-84.

9. Virchow, R., cited by Roberts.[10]

10. Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, Vol. 3, p. 968.

11. Kettel, K.: Neurinoma of the Facial Nerve, Arch. Otolaryng. 44:253-260 (Sept.) 1946.

12. Tarlov, I. M.: Origin of the Perineural Fibroblastoma, Am. J. Path. 16:33-40 (Jan.) 1940.

13. Penfield, W.: Tumors of the Sheaths of the Nervous System, in Penfield,[10] pp. 955-990.

14. Mallory, F. B.: Type of Cell of So-Called Dural Endothelioma, J. M. Research 41:349 (March) 1920.

15. Ewing, J.: Neoplastic Diseases, Ed. 4, Philadelphia, W. B. Saunders Company, 1940.

16. Roberts, G. J.: Neurinoma of the Facial Nerve in the Middle Ear and Mastoid: Report of a Case, Arch. Otolaryng. 37:62-73 (Jan.) 1943.

17. Williams, H. L., and Pastore, P. N.: Neurofibroma of Facial Nerve in Facial Canal with Destruction of Labyrinth and Mastoid Process, Arch. Otolaryng. 29:977-981 (June) 1939.

18. Bogdasarian, R. M.: Neurinoma of the Facial Nerve, Arch. Otolaryng. 40:291-294 (Oct.) 1944.

19. Skoog, T., cited by Kettel.[11]

20. Lundgren, N.: Neurinoma n. facialis, Acta oto-laryng. 35:535-537, 1947. (not read)

21. Loeliger, H. T.: Über Fazialisneurinome, Acta oto-laryng. 35:543-555, 1947. (not read)

22. Rainer, V.: Un cas de neurinome du nerf facial, Ann. oto-laryng. 71:770-778, 1954. (translation)

23. Schmid, C., cited by Kettel.[11]

24. Ehrlich, H. E., and Martin, H.: Schwannomas (Neurilemomas) in the Head and Neck, Surg. Gynec. & Obst. 76:577-583 (May) 1943.

25. Ballance, C., and Duel, A. B.: The Operative Treatment of Facial Palsy, Arch. Otolaryng. 15: 1-70 (Jan.) 1932.

True Cholesteatoma

A Report of Two Cases

JAMES L. SHEEHY, M.D., Los Angeles

A true cholesteatoma of the temporal bone is a rare tumor, derived from a congenital epidermal rest.[1-6] This is in marked contrast to the rather common pseudocholesteatoma associated with a marginal perforation of the tympanic membrane and chronic aural suppuration.[1,3,6,7] During the last year and a half two cases of apparent true cholesteatoma have been treated. One presented itself as a middle-ear tumor, the other as an idiopathic facial nerve paralysis of seven months' duration.

CASE 1.—An 8-year-old boy was first seen in March, 1956, because of left ear pain, for one day, three weeks previously. He had had an earache two years before without otorrhea, but no other ear trouble. A hearing loss had not been noted.

Examination revealed a boy in excellent health with a normal nose, throat, and right ear. There was bulging of the pars flaccida and posterior superior quadrant of the left tympanic membrane, and slight sagging of the posterior superior canal wall. No evidence of an acute inflammation or discharge could be found. The anterior portions of the tympanic membrane appeared normal. X-rays showed a diploic left, and a normal right mastoid. There was a 32 db. conductive hearing loss.

Because of the lack of symptoms, and some question as to the activity of the disease process, the ear was observed for two months. There was no change. On May 7 the left mastoid was explored endaurally. The antrum was free of disease. Granulation tissue surrounded the incus, and a small cholesteatoma was found in the posterior superior portion of the middle ear. This had destroyed the head and neck of the stapes and a portion of the long crus of the incus, and had worked its way into the bone beneath the pyramidal segment of the facial nerve. All matrix was removed, and a radical operation was performed.

The postoperative course was uneventful, and healing was taking place satisfactorily when the patient was last seen, five weeks postoperatively. He failed to return for further observation.

Comment.—There was, in retrospect, no indication for the two-month period of observation; a tumor obviously existed and required surgery.

CASE 2.—A 25-year-old Negro was referred in September, 1957, for evaluation of a right facial nerve paralysis of seven months' duration. The paralysis had developed gradually over a two-week period without other symptoms. The patient believed that there may have been some fluctuation in the "feeling" in his face, but stated that he was unable to whistle or close his eye completely from the time of onset.

No treatment was instituted until four months after the onset of the paralysis, at which time it was described as complete. Cortisone and physical therapy were started, but were discontinued after one month. Five and a half months after the onset of paralysis the patient developed severe pain in the right ear. Over a period of the next month and a half this pain recurred at intervals, and was associated with the onset of a mild hearing loss. At no time had there been otorrhea or vertigo.

The patient had always been in good health, having had no ear, nose, or throat disease. He did not remember earache or aural discharge in the past. (Correspondence later with his parents confirmed the lack of otitis media in childhood.) On rare occasions over the past year he had become mildly unsteady upon sudden change of posture.

Examination revealed a healthy adult with a complete right facial nerve paralysis. There was poor muscle tone. Taste sensation was absent on the anterior two-thirds of the tongue on that side, but the tearing reflex was present. The nose, throat, and left ear were normal. There was obliteration of the landmarks of the upper posterior portion of the right tympanic membrane, with a suggestion of fullness of the posterior superior canal wall. The posterior ligament and short process could be brought into relief by increase in pressure in the canal with the Bruening otoscope. The tympanic membrane showed no evidence of perforation or scarring. Weber and Rinne tests indicated a conductive loss of hearing

Submitted for publication April 4, 1958.

Formerly Major, Medical Corps, U. S. Army.

Fig. 1 (Case 2).—Towne view showing large area of radiolucency in the right mastoid.

Fig. 2 (Case 2).—Posterior semicircular canal with curved needle through the lumen.

on the right, and audiogram showed a 32 db. loss. The response to cold caloric stimulation of the horizontal semicircular canal was markedly diminished, but present, nonetheless.

X-ray examination (Fig. 1) demonstrated an acellular right mastoid with a 1×3 cm. radiolucent area involving the antral, tip, and supralabyrinthine area, with a suggestion of involvement of the internal auditory canal. Electrodiagnostic tests of the right facial musculature showed little evidence of fibrosis, suggesting that reinnervation would result in satisfactory function. There was no evidence of voluntary motion.

On Sept. 19 a right radical mastoidectomy was performed endaurally. A large cholesteatoma was uncovered, packed solidly with keratin. There was no evidence of infection, and no evidence of connection with either the Eustachian tube or the external canal. The tumor involved the mastoid process to the level of the floor of the canal, and exposed the sigmoid sinus in a small area. The retrolabyrinthine area had been invaded, and the endolymphatic sac uncovered. The tumor had grown inside the loop formed by the posterior semicircular canal and had fractured it loose from the capsule of the inner ear (Fig. 2). The superior vertical canal had been destroyed, and the roof was missing from both the vestibule and the internal auditory canal. The dura was exposed by the disease immediately above the internal canal. The incus and stapes were missing. The facial nerve had been exposed from the midportion of the pyramidal segment, to a point 3 mm. within the internal auditory canal. In the tympanic segment all but a small fraction of the circumference of the nerve was covered by squamous epithelium, and the nerve could be freely moved with a probe. The malleus and tympanic membrane were removed, revealing a normal middle ear below the level of the fossa ovalis. Matrix was left in place where it covered the dura, vestibule, internal canal, and facial nerve.

Postoperatively there was mild vertigo for three days. Packing was removed on the fourth postoperative day, and there was practically no otorrhea following this. A dry treatment was used (sulfonamide-chloramphenicol powder) intermittently, and the cavity healed without incident in nine weeks. Return of facial nerve function was first noted in the orbicularis oculi (three weeks), followed by the frontalis (five weeks) and orbicularis oris (eight weeks). Audiogram showed a 50 db. conductive hearing loss.

Histological examination of tissue removed at surgery showed the usual picture of cholesteatoma. There was, however, no evidence of infection or of cholesterol clefts.

Comment.—The matrix was left in place, where it covered the facial nerve, horizontal canal, roof of the vestibule, internal auditory canal, and dura. This was done because it was felt that there would be less likelihood of damage to the nerve and vestibule, of the production of a cerebrospinal fluid otorrhea, or of later occurrence of secondary cholesteatoma. Although there are some [4] who might criticize this decision, others [3,7] utilize the matrix regularly in selected cases without regret.

This case presented many interesting findings. There was destruction of a large portion of the temporal bone, explainable only on a basis of pressure, without pain until late in the disease. Extensive destruction of the vestibular labyrinth occurred without causing more than rare episodes of postural vertigo. The hardness of the capsule of the inner ear is amply demonstrated here: The tumor, approaching the posterior

semicircular canal, worked its way inside the loop formed by the canal, fracturing it loose, without damage to the hard bone of the canal itself (Fig. 2). Normal response of the facial musculature to galvanic stimulation was quite a surprise. This, along with the patient's statement of a fluctuation in the "dead" feeling in the face, was interpreted as indicative of an intermittent, or incomplete, block in the facial nerve. The early return of function after surgery substantiated this.

General Comment

Day [2] and House [8] reported cases similar to Case 1. Day's patient had a true cholesteatoma which had disrupted the incudostapedial joint. An atticotympanotomy was performed, with complete removal of the cholesteatoma. After the incus and malleus head had been removed, a tympanoplasty was accomplished. House's patient complained of vertigo and had a 30 db. conductive hearing loss. Mastoidectomy revealed a small unsuspected cholesteatoma in the fossa ovalis. The stapes had been destroyed. A radical mastoidectomy was performed.

Seven cases of apparent true cholesteatoma similar to Case 2 have been reported. Jefferson and Smalley [9] reported six cases of progressive facial palsy due to noninfected petromastoid epidermoids. These were thought at first to be cases of idiopathic palsy (as in my case). Surgery demonstrated benign epidermoids of congenital origin. Tamari's case [6] of apparent true cholesteatoma presented itself as an idiopathic facial nerve paralysis of one and a half years' duration. Massive temporal bone destruction was found at surgery. In light of the experience with these seven cases and my own case, it should again be emphasized that temporal bone radiography must not be omitted in any case of apparent idiopathic facial-nerve paralysis, despite lack of ear symptoms.

The diagnosis of true (congenital) cholesteatoma is based primarily on the lack of history of otorrhea in a patient with a benign noninfected temporal bone epidermoid having no connection (perforation) with the external auditory canal. Diamant,[10] however, believes that the cholesteatoma always precedes the perforation and that the aural cholesteatoma may be a true tumor. Ruedi [11] also believes that the aural cholesteatoma may develop behind an intact tympanic membrane. The cases presented and referred to here are interesting and unusual, but one wonders whether a diagnosis of *true* cholesteatoma would have been made had a perforation and otorrhea developed previous to observation by an otologist.

Summary

Two cases of apparent true cholesteatoma of the temporal bone are presented. One case presented itself as a middle-ear tumor, the other as a facial-nerve paralysis of seven months' duration.

1136 W. Sixth St. (17).

REFERENCES

1. Eggston, A. E., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, The Williams & Wilkins Company, 1947.

2. Day, K. A.: A Primary Cholesteatoma of the Middle Ear, Ann. Otol. Rhin. & Laryng. 64:161-163 (March) 1955.

3. Juers, A. L.: Modified Radical Mastoidectomy, A. M. A. Arch. Otolaryng. 57:245-256 (March) 1953.

4. Walsh, T. E.; Covell, W. P., and Ogura, J. H.: The Effect of Cholesteatosis on Bone, Ann. Otol. Rhin. & Laryng. 60:1100-1113 (Dec.) 1951.

5. Begley, J. W.; McDonald, J. R., and Williams, H. L.: Histology and Histogenesis of Cholesteatoma of the Middle Ear and Mastoid, A. M. A. Arch. Otolaryng. 53:41-52 (Jan.) 1951.

6. Tamari, M. J., and Fogo, A. E.: True Cholesteatoma of the Temporal Bone, A. M. A. Arch. Otolaryng. 54:518-523 (Nov.) 1951.

7. Baron, S. H.: Modified Radical Mastoidectomy, Arch. Otolaryng. 49:280-302 (March) 1949.

8. House, H. P.: An Apparent Primary Cholesteatoma: Case Report, Laryngoscope 63:712-713 (Aug.) 1953.

9. Jefferson, G., and Smalley, A. A.: Progressive Facial Palsy Produced by Intratemporal Epidermoids, J. Laryng. & Otol. 53:417 (July) 1938; cited by McGovern, F. H., and Fitz-Hugh, G. S.: Diseases of the Facial Nerve, Laryngoscope 66: 187-236 (March) 1956, p. 225.

10. Diamant, M.: Anatomical Etiological Factors in Chronic Middle Ear Discharge, Ann. Otol. Rhin. & Laryng. 66:380-389 (June) 1957.

11. Ruedi, L.: Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone, Ann. Otol. Rhin. & Laryng. 66: 283-305 (June) 1957.

Voice Therapy

Part II. Essential Treatment Phases of Specific Laryngeal Muscle Dysfunction

F. BERTRAM BRIESS, Syracuse, N. Y.

In Part I, I discussed physiology of the larynx during phonation; voice testing, classification, and diagnosis.[1] This second paper describes the specific therapeutic approach used for the restoration of normal laryngeal muscle balance, as currently used in the Department of Otolaryngology at the State University of New York, Upstate Medical Center at Syracuse.

To be fully effective, voice therapy must restore the normal dynamic equilibrium of the intrinsic and cricothyroid muscles which control the functions of the vocal cords. Further, the patient must learn how to maintain this equilibrium. This requires specific, thorough, and precise adjustment of these muscles. Voice therapy naturally divides itself into four distinct but closely interrelated phases:

1. The first, or Dehabiting Phase, during which voice habits indicative of abuse are identified and corrected

2. The Muscle Retraining Phase during which correction.

3 & 4. Widening of safety margin, by training all muscles to endure far more than the untrained voice can possibly withstand, and education of the patient to feel the laryngeal sensations associated with beginning muscle imbalance, and to know how to accomplish immediate correction.

Phase: Dehabiting

During this phase, the patient is instructed how to avoid damaging phonation errors which are unwittingly developed, as the dynamic equilibrium is upset through such abuse. It may be remembered that this procedure is an essential preliminary step before successful testing may be accomplished. Elimination of these same abuse habits therefore becomes the first obligation of the therapist. The following illustrative examples are again listed:

A. Abusive breathing habits.
   1. Inspiration through partially closed cords (unhealthy inspiration).
   2. Excessive intake of air—too much air too often.
   3. Glottal attacks—excessive, forceful posterior closure of the cords heard at the beginning of syllables.
   4. Breathy voice stemming from bowed vocal cords. This can be quickly repaired within seconds and represents hyperfunction of the posterior cricoarytenoid, or on occasion interarytenoid muscles, as seen in untrained or partially trained voices, or on coughing or throat clearing.
   5. Excessive air escape caused by edema or lesions on the cords. The patient develops a habit by continually trying to compensate more and more for the waste of air due to the edema or lesion.

B. Pitch adjustments—too low pitch with too little or too much volume as in pseudoauthority or in psychosomatic overlay cases.

C. Sound distortion representing abusive exaggerated muscle response to mirth, sadness, etc.

D. Whisper-like phonation in patients working in hospitals, libraries, and places where normal volume of voice is shunned.

Each of these habits cannot be dealt with singly and corrected completely before therapy of specific muscles results in voice improvement. In fact, voice may temporarily become less clear and weaker while the above habits are being corrected.

Such abnormal phonation may have its start when voice is overused during laryngitis (secondary to upper respiratory infection), or during menstruation, or it may stem from continued use through the delicate mutation period.

Submitted for publication May 14, 1958.

Instructor, Department of Otolaryngology, State University of New York, Upstate Medical Center at Syracuse.

A large percentage of voice disorders among industrial workers, especially in this country, have been observed to result from voice use over machinery noise. Voice disorders in trained speakers or singers usually result from incorrect voice methods. Such patients must be helped to eliminate habits which they acquired through such training.

Psychogenic factors usually intensify existing muscle imbalance. The symptoms associated with the muscle imbalance, when quickly relieved by the voice therapist, reassure the patient. In certain instances emotional tensions are of such magnitude that professional psychiatric consultation is necessary. Yet, in a very large percentage of patients personal and social problems as handled by the otolaryngologist-therapist team resolve satisfactorily.

The patient should be informed about the mechanics of voice production. He must also be made aware of the damaging effects of each of the abusive habits. Moreover, improved voice quality and relief of throat symptoms, even though temporary at first, is accomplished within the first session. By doing so the therapist wins the patient's confidence while the latter's self-confidence returns.

Phase II

The second therapy phase represents the main stage and is concerned with retraining, namely, lessening the tension of the hyperfunctioning pairs and tensing or reactivating the antagonists. Duration of this procedure depends on the pathology of the lesion or condition and on rapidity of response of the involved muscles to treatment.

To this end, there is a definite need for understanding individual phonatory sounds which determine the action of each specific muscle pair. These sounds are directly dependent upon the following factors: (*a*) choice of vowel; (*b*) pitch; (*c*) subglottic compression; (*d*) phrase design; (*e*) dosage.

Utilizing the speaking voice, work phrases are chosen which employ the specific vowel or vowels or vowel assimilations which will improve the function of the indicated muscle pair. The order in which muscle pairs are treated depends on results of testing and retesting to detect existing or developing hyper- or hypofunctions. Type and character of voice, as well as nature of lesion, dictate the choice of vowel.

Choice of Vowel.—English vowels, a, e, i, o, u; their assimilations, as, ŏ, ō, ô, o͝o, ŭ, ă, ā, â, ä, ĕ, ē, ī, o͞o, o͝o, ū; French and German vowels; double vowels, diphthongs, as well as sequential vowels ā to ū, are all employed. Change of one vowel into a different vowel is made possible by a sudden increase in activity of one pair of muscles and a corresponding and proportional decrease in activity of its antagonist.

As early as 1911, Pielke[2,3] referred to "covering of the tone" and observed changes in the position of the arytenoids and epiglottis during phonation when vowels were changed. Iro[4] explains in great detail the physiology of the larynx during vowel production. He states that any assimilation to the vowel "o͞o" (as in "food"), or darkening of any vowel "ā" (as in "lake" into "û" as in "fur"), results in increase of "free-edge vibration." This procedure, which is called "covering of the tone," is accomplished through lowering of the larynx and consequent elongation of the vocal cords.

Each vowel, depending on the specific voice type and voice character as well as specific pitch and degree of subglottic compression, produces a definite and predictable change in the individual muscle balances. The muscular change required in shifting from one vowel to another is the crux of this therapeutic approach.

The following facts are characteristic for high female voices: (1) occurrence pitch of true divergence above e_2; (2) fast (sudden) diminishing of full vibration function below c_1; (3) best (least abusive and most comfortable) phonation between c_2 and d_2. The facts that the patient's voice cannot endure "accenting" (sudden at-

tacks) on other than a (as in "lake") without further disturbing the thyroarytenoid cricothyroid function, plus the fact that true divergence occurs not lower than at f_2 sharp prove the delicate character of 75 to 90 degrees.

Here is an example: A female patient with a 0.5 mm. firm node on the right cord at the junction of the anterior and middle thirds. Testing reveals a high type voice of delicate character. The patient phonates and sings with extreme hyperfunction of cricothyroids, together with hyperfunctioning lateral cricoarytenoid muscles.

Ō vowels (as in "soul") and/or ue (German umlaut ü) are used in the region of occurrence pitch of divergence (between f_2 sharp and g_2), because these vowels improve function of the antagonistic thyroarytenoids.

OO (as in "food") and oo (as in "full") are used in words and phrases beginning six to eight tones below the divergence occurrence pitch, and pitch of such phrases is gradually raised towards divergence occurrence pitch.

Pitch.—Since degree of contraction of all participating muscles changes when contraction ratios of one of these muscle pairs is altered, it is suggested that this equilibrium be therefore termed *dynamic*. This equilibrium varies directly with the frequency of the tone being produced. Therefore, phrase patterns are designed in such a manner that words or syllables which contain specific vowels are requested at specific frequencies. Choice of frequency depends on type, character, and exact muscle pair being treated (see Therapy, Phase II).

Change of pitch, while a specific vowel is produced, affects momentarily the thyroarytenoid cricothyroid muscle balance, while change of a specific vowel at a sustained pitch alters the cricoarytenoid balance and, consequently, posterior closure.

Local or "Pseudo" Divergences: In voices with distorted equilibrium, so-called "local divergences" may show at any pitch above the lower third of the voice compass. This pitch is variable in a particular voice, and

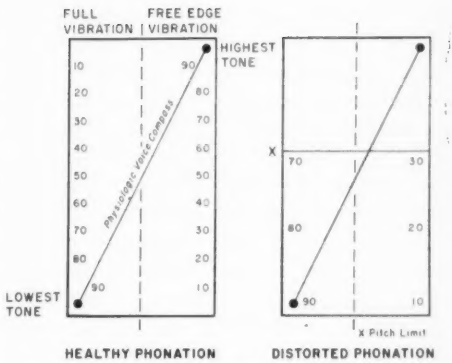


Fig. 1.—In healthy phonation, the lowest tone of the natural voice compass may consist of 90% thyroarytenoid and 90% cricothyroid function, while the highest tone may be compounded of 10% thyroarytenoid and 10% cricothyroid function.

depends entirely on type of abuse and momentary degree of hyperfunction of fatigue of a specific muscle or muscle pair. Such pseudodivergences are frequently caused (1) in robust character voices by excessive hyperfunction of thyroarytenoids in the presence of undercompression, and (2) in delicate character voices by hyperfunction of cricothyroids in the presence of subglottic overcompression. There are borderline cases where subglottic over- and undercompression alternate within a specific phrase. Caution is indicated when classifying such a voice, for these reasons, as occurrence pitch or true divergence may be confused with pitch of pseudodivergence (Figs. 1 and 2).

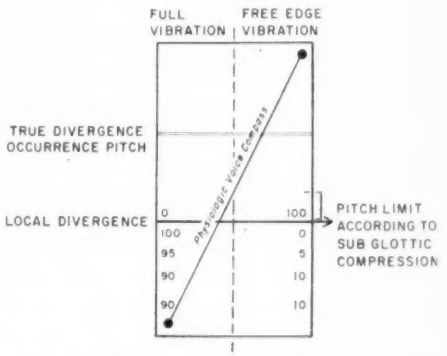


Fig. 2.—Hyperfunction of thyroarytenoids or/and hypofunction of cricothyroids results in limited pitch.

Subglottic Compression.—Well-balanced subglottic compression is the third important factor in healthy phonation.

Subglottic compression is variable within a particular phrase. A specific vowel, at constant pitch and volume is normally produced under stronger (higher) subglottic compression at the beginning of a phrase, under less subglottic compression, if the vowel is placed in the middle of the same phrase, and lowest at the end of the phrase, when air supply (breath) is at the lowest. Trained speakers or singers, as well as patients with voice disorders may prove otherwise, depending on the acquired breathing technique, respectively dependent on condition of voice or lesion.

The power to "hold a long breath" is unimportant in phonation, as vital as it might be for diving and other sports. On the contrary, it is essential with phonation to refrain from inhaling too much air because of the danger of inflexibility of abdominal muscles. Unfortunately, breathing is one part of speech and voice methods where the greatest blunders are made. Explosive and compressive breathing dangerously affects dynamic equilibrium of the laryngeal muscles. Constant pressure of restrained air, regardless of volume and pitch, usually causes gradual loss of "free-edge vibration." Such abuse frequently produces a function divergence in voices belonging to the robust character groups, or will result in hyperfunction of cricothyroids in delicate character voices.

Subglottic compression depends primarily on tension and shape of the vocal cords, as well as closing and opening time. Findings of recent research conducted by Van Den Berg, Zantema, and Doornenbal, Jr.[5] reaffirm these facts and prove the variability of subglottic compression during phonation.

Subglottic overcompression distorts not only the interplay of thyroarytenoid cricothyroid muscles; it also causes hyperfunction of interarytenoid muscles.

Lack of subglottic compression may result in hypofunction of thyroarytenoids, and in extreme cases dysphonia plicae laryngis may develop when the patient subconsciously attempts to compensate for the decrease of tone volume by substituting the false vocal cords. Extreme hyperfunction of the lateral cricoarytenoid muscles always accompanies such abuse, and the false cords approximate on attempted phonation.

Phrase Design.—The preceding three factors—choice of vowel, pitch, and subglottic compression—all are simultaneously utilized in therapy in the procedure herein termed phrase design. As further described in succeeding sections dealing with the specific therapy of individual muscle pairs, not only the choice of vowel but the pitch at which it is produced and the subglottic compression with which it is produced as well as the shift from one vowel form to another produce the strengthening of a particular muscle pair. Figure 3 indicates shift of vowel form affected by pitch and subglottic compression. A wide variety of vowel shifts, pitches, and lengths of phrases must be designed literally "on the spot" as the therapist molds and adjusts the balance of the involved muscles. A single phrase may need to be repeated as many as 50 or 100 times at the correctly determined pitch

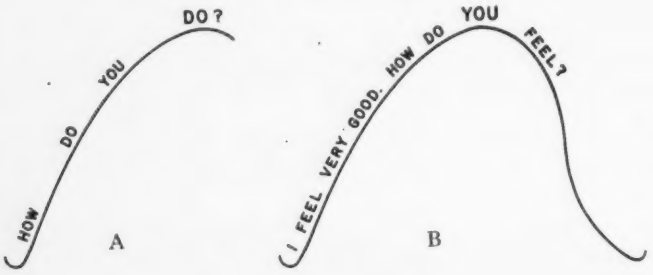


Fig. 3.—*A*, phrase pattern. *B*, extended phrase pattern.

and subglottic compression to produce the desired improvement in function. This at once leads us to a consideration of dosage.

Dosage.—The muscles which control the functions of the vocal cords are very delicate and are easily irritated. They can be stimulated or relaxed within a few minutes. They respond promptly to treatment. If benign lesions are present, the changes grossly alter vibration of the cords. Response is definite but slower; yet there is progressive improvement as the lesion disappears by this mode of treatment.

Correct dosage is of vital importance. Here again it will depend largely on voice type and character, how soon and to what extent overdosage could cause damage.

Adjustment of function balances may clear vocal cords of newly formed nodes within three to four sessions, while the patient carries on regular professional work. However, there are also cases recorded where overdosage of a specific treatment resulted in a new and sometimes different lesion, although the original lesion had disappeared. Such newly produced lesions can be successfully treated if recognized in time at periodical check-ups with the discerning laryngologist. If neglected, however, the altered production (phonation) will develop into a different (new) type of abuse and cause a different abnormality.

The therapist also anticipates that some overanxious or overzealous patients will actually overdose themselves by exaggerating specific treatment procedures. Therefore, subsequent treatments may have to be altered, in order to relax muscles which had been hypofunctioning originally.

Hyperfunction of Thyroarytenoids.—Balance of thyroarytenoid and cricothyroid functions is distorted when thyroarytenoids are hyper- or hypofunctioning. Full vibration and free-edge vibration are out of proportion; moreover, this imbalance becomes severer when pitch is changed. Ratios of both functions adjust automatically in a healthy voice. Figure 1 shows the principle of the changing balance in a healthy voice when pitch is raised.

Figure 1 also illustrates incorrect proportion of functions in a voice belonging to the same type and character when hyperfunction of thyroarytenoids exists. Thyroarytenoid muscles fail to relax gradually when pitch is raised. Such dysfunction results in pitch limitation, and paretic hoarseness may develop when thyroarytenoids fatigue.

The therapist has two alternative approaches when dealing with such a condition, namely, relaxation of thyroarytenoid muscles or tensing of cricothyroids. The particular functioning characteristics of a specific voice type and character dictate in most cases the procedure to be used.

Reduction of the hyperfunction of the thyroarytenoids cannot be achieved while pitch is raised unless subglottic compression is decreased at the same time.

Phrases with a major number of words which contain "u" ($\bar{o}\bar{o}$ as in "food") vowels are used in ascending pitch patterns at medium volume. The patient must be trained to widen his speaking range upward (Fig. 3*A*). Subglottic compression is inclined to increase when the voice is reaching higher pitches under existing hyperfunction. Longer phrases should be used (Fig. 3*B*), and intensity must be increased in order to counterbalance overdeveloping subglottic compression. Local divergences may lead to misclassification, a mistake with far-reaching consequences (Fig. 1).

Hypofunction of Thyroarytenoids.—Phrase patterns designed for the treatment of hypofunctioning thyroarytenoids omit words with "u" (oo) vowels or assimilations. Sounds such as the French eu, or the German "ö" (Umlaut o) or û (as in "Sir") are used, in phrases which must not exceed one octave to one none (nine tones) of the medium part of the patient's physiologic voice compass. The patient ought to be instructed to start phrases at his most comfortable speaking level, where the least breathiness is evident. Only ascending patterns should be used which at no time approach divergence occurrence pitch more closely than by two (musical) steps (one

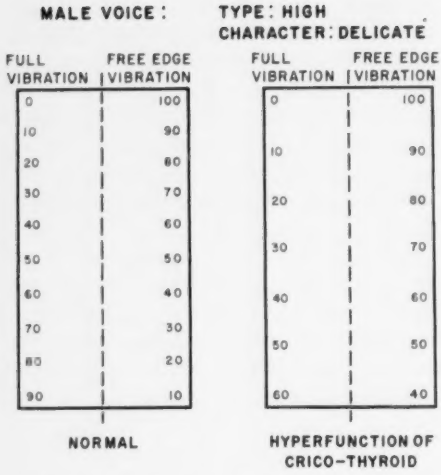


Fig. 4.—Proportion of full vibration and free-edged vibration in normal phonation and in cricothyroid hyperfunction.

and one-half tones in the minor scales or two full tones in the major).

Subglottic compression must be carefully adjusted to pitch changes. Undercompression increases breathiness of tone while overcompression aggravates the patient's sensation of presence of a lump below the cricoid.

Pseudodivergences occur frequently very close to "true" divergence occurrence pitch, and it might be impossible for the therapist to quickly distinguish between true and pseudodivergence. In such instances he is compelled to judge functioning condition on the basis of sensations in the patient's throat. Degree of "lump feeling" which usually is present in patients with hypofunctioning thyroarytenoids indicates progress of treatment.

Cricothyroid Hyperfunction.—Hyperfunction of cricothyroid muscles during phonation is responsible for predominence of free-edge vibration in the patient's voice (Fig. 4). Such imbalance is found more frequently in women than in men.

The treatment is similar to the procedure described for hypofunction of thyroarytenoids with the exception of pitch arrangement. Phrase patterns must cover most of the physiologic voice compass. Treatment ought to concentrate in the vicinity of occurrence pitch with the goal of elimination of divergence. An acute or chronic function divergence is usually present in voices with hyperfunctioning cricothyroid muscles (Fig. 5). Use of descending patterns is prohibited for the duration of the disturbance because such phrases provoke pseudodivergence at unfavorable pitch levels, especially in female voices.

Hypofunction of Cricothyroids.—Voices usually lack free-edge vibration when hypofunction of cricothyroids exists. Thera-

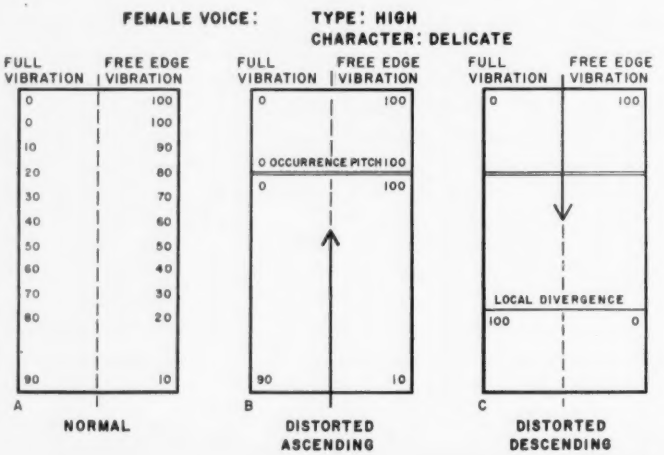


Fig. 5.—*A*, proportion of full vibration and free-edge vibration in normal phonation. *B*, phonation with hyperfunctioning cricothyroids shows divergence at the actual occurrence pitch in ascending phrases. *C*, in descending phrases, however, divergence may occur anywhere within the lower half of the compass in the same voice.

peutic treatment with the object of regaining this vital function may take up considerable time. Phrases composed of words with "oo" (u) vowels and assimilations are utilized in ascending and descending patterns at modest or minimum volume, especially in the region of divergence occurrence pitch. No function divergence exists in cases of extreme hypofunction of cricothyroids. Therefore, classification is impossible as long as occurrence pitch cannot be established. In the course of the treatment flageolet tones occur in the region of (the true) occurrence pitch and later a function divergence develops which proves some recovery of free-edge vibration. Dosage of treatment must be carefully watched. In case of overdosage, the patient complains about pain, which usually starts in the region of the arytenoids and moves upward and backward toward the ears. In such event the therapist ought to change the course of treatment immediately. However, the patient must not be allowed to rest; on the contrary, the therapist will counteract the overtreatment with a procedure as described for hypofunctioning thyroarytenoid muscles.

Cricoarytenoid Muscles.—Dysfunction of cricoarytenoids is based on hyper- or hypofunction of either lateral or posterior cricoarytenoid muscles. The inferior functioning condition may be uni- or bilateral with or without visible paresis or paralysis.

Hyperfunction of Lateral Cricoarytenoids.—Hyperfunction of lateral cricoarytenoids results in breathy and weak voice quality. The patient develops abusive breathing habits because of shortness of breath due to poor posterior closure. Imbalance of thyroarytenoid cricothyroid muscles is always found to be extreme when hyperfunction of lateral cricoarytenoids exists. Cricothyroids are usually hyperfunctioning in voices belonging to medium or high types and delicate characters. Hypofunction of thyroarytenoids shows in voices of low type and robust character groups.

Adjustment of thyroarytenoid and cricothyroid muscles must precede treatment of cricoarytenoids. Therapy of cricoarytenoids is complicated by the fact that thyroarytenoid-cricothyroid balance changes when pitch or vowel are altered. Correct cricoarytenoid balance depends on the momentary interplay of those muscles, as well as on changes in subglottic compression.

Treatment aimed at correction of thyroarytenoid or cricothyroid dysfunction and successful treatment of lateral cricoarytenoids improves voice quality. The patient often overdoses one specific treatment and fluctuates between hyper- and hypofunction of a particular muscle. Lump feeling and side pain (in the region of the arytenoids) alternate according to the momentary functioning condition. Frequently, these shifting sensations are important indications for the therapist as to progress and direction of treatment.

Endurance and divergence tests are applied repeatedly in order to determine response of thyroarytenoid and cricothyroid muscles to treatment. Pseudodivergencies usually show at varying frequencies above the actual divergence occurrence pitch, especially when the patient attempts to increase intensity of tone. As soon as dysfunction of thyroarytenoids and cricothyroids is sufficiently corrected, that is, when pseudodivergencies appear at least three tones below actual divergence occurrence pitch, the patient is instructed to increase subglottic compression. Patients with hyperfunctioning lateral cricoarytenoids are unable to produce round "o" vowels (as in "go"). The French diphthong "eu" is used in ascending patterns. Phrases should be kept short, and should cover at least 10 musical steps, overstepping divergence occurrence pitch by one or two tones. Tone volume must never be below medium loud. Failure to adjust subglottic compression at the same time leads to increased lump feeling, if thyroarytenoids do not respond, or when fatigue sets in. In such instances, phrases should be lengthened and pitch ought to be limited in such manner that the highest pitch of the patterns used is kept at least one tone below divergence occur-

rence pitch. Thyroarytenoid-cricothyroid interplay is tested repeatedly during treatment, and readjustment to altered cricoarytenoid balance is made whenever necessary.

Hypofunction of Lateral Cricoarytenoids. Hypofunction of lateral cricoarytenoids is a consequence which occurs in voices with hyperfunctioning posterior cricoarytenoids. Treatments are identical (see below).

Posterior Cricoarytenoids.—Reduction of subglottic compression and elimination of glottal attacks are the major aims of voice therapy in such cases where hyperfunction of posterior cricoarytenoids exists. Again thyroarytenoids which are usually hyperfunctioning in such cases are treated first. The use of a wider range is always indicated; therefore, phrases with "oo" and o vowels are used with little volume. Long phrases of 10 to 20 words which contain such vowels help to decrease subglottic compression.

Hypofunction of posterior cricoarytenoids is in most cases based on hyperfunction of their antagonists. Treatment is aimed at lessening the existing hyperfunction of lateral cricoarytenoids (see above).

In certain instances, hypofunction of posterior cricoarytenoids stems from an existing hyperfunction of the interarytenoid muscles. Such malfunction usually leads to aphonia. Restoration of normal function is only possible after relaxation of interarytenoid muscles. The patient is instructed how to refrain from excessive throat clearing; he is taught how to cough and giggle with a minimum of interarytenoid action. Subglottic overcompression and hectic attempts at the beginning of phonation are sometimes remedied by gurgling or similar exercises which are designed to recover normal function of posterior cricoarytenoids.

Phase III & IV

When the dynamic equilibrium is restored, the original voice difficulties disappear. The patient's voice quality shows obvious improvement and the patient phonates with ease. However, abusive habits may return, when the patient is discharged too soon. He is liable to become careless, without supervision, and a serious relapse may occur when he overuses his voice again with overconfidence. During the third therapy phase the patient continues practice of the newly acquired phonation rules until voice production becomes nearly automatic. In other words, during the third therapy stage, the patient is trained to observe subconsciously the regulations which keep his voice healthy.

However, in most cases, even such precautions will not guarantee permanent improvement unless all muscles are trained to endure more than perfect use of the voice demands. Brewer[6] points out that vocal cords can withstand considerable abuse, depending upon the state of dynamic equilibrium. However, when the range of tolerance has been exceeded, then definite symptoms and signs of vocal abuse are evident. Brewer also emphasizes the fact that this range is very wide in trained voices and very narrow in untrained voices.

It is practically impossible under tensions of present-day living for anyone to refrain from overuse and frequent abuse of the voice. Not only professional speakers, ministers, teachers, lawyers, but also salesmen promoting a new product, or factory workers under loud machinery noise, telephone operators, and plane pilots, as well as mothers in order to keep their children in line, overstep that natural safety margin every day.

For this reason, no therapeutic treatment can be considered complete without the fourth therapy phase which is used to widen the abuse tolerance. All muscles must be "overtreated" and trained to endure more than normal voice production demands. The patient must also learn to feel malfunction of muscles in his larynx due to abuse or emotional factors. He must know how to readjust the vocal instrument long before voice quality becomes seriously affected.

Vocal cords of opera singers who sing continuously for several hours, using their voice over an extremely extended range, and with exceptionally loud volume, in large halls over an orchestra of 100 to 140 musicians, will show nothing worse than redness of the vocal cords. The experienced laryngologist accepts such a condition after a performance as normal for the individual. He knows that especially thyroarytenoids of a singer's throat are overtrained and can endure abuse which would produce edema, nodes, polyps, or a more serious lesion in an untrained voice.

Comment

From the above description of the four therapy phases, with special emphasis on Phase II it becomes evident that the therapist must obtain the necessary musical background, for he must know without hesitation the exact pitch of each essential vowel as it is produced by the patient in words and phrases. This requires accurate and rapid pitch identification, as well as the ability to transpose immediately to a higher or lower pitch with reference to the divergence occurrence pitch, or to redesign the entire phrase and personally illustrate or direct the patient in how to accomplish the desired result. Absolute pitch is desirable but not essential. However, a 440 tuning fork as a reference level, or, for the less gifted, a piano or organ will suffice.

Summary

A further appreciation of the four phases of voice therapy necessary for each patient has been presented for the interested otolaryngologist. The elementary steps in determining choice of vowel, pitch, management of subglottic compression, phrase design, and handling of dosage are explained. An outline of the mechanics of treating the intrinsic and cricothyroid muscles of the larynx is briefly presented. It becomes evident that because of rapidity of response of the involved muscles to the above-described stimuli that oversimplification of the therapy procedure is dangerous. Just as in the field of medicine, an article or even a textbook, though stimulating, must be supplemented by supervised training in connection with clinical observation to be safe and effective. Even though the otolaryngologist lacks the extensive musical background and can ill afford the time personally to instruct his patients in this specialized type of treatment, yet with background material such as has been presented, he has sufficient knowledge to guide his patient. Proper selection of a voice therapist and interval indirect laryngoscopy and otolaryngological examination to guide the therapist will provide essentials for successful treatment of the patient.

State University of New York, Upstate Medical Center at Syracuse.

REFERENCES

1. Briess, F. B.: Voice Therapy: Part I.: Identification of Specific Laryngeal Muscle Dysfunction by Voice Testing, A. M. A. Arch. Otolaryng. 66:375-382 (Oct.) 1957.

2. Pielke, W.: Über "offen" und "gedeckt" gesungene Vokale, Paasow-Schafer Beitr. 5:215, 1911.

3. Pielke, W.: Über den Ausgleich des Stimmbruchs durch die sogenannte Deckung, presented at the Third International Laryngological Congress, Berlin, 1911.

4. Iro, O.: Diagnosis of the Voice, Die Stimmbildung, Vienna, 1923, pp. 67-68, 1957.

5. Van Den Berg, J. W.; Zantema, J. T., and Doornenbal, P., Jr.: On the Air Resistance and the Bernoulli Effect of the Human Larynx, J. Acoustical Soc. America 29:626-631, 1957.

6. Brewer, D. W.: Medical Aspects of Voice Therapy, Bull. Nat. A. Teachers of Singing 9:4-7 (March-April) 1953.

Tympanic Skin Grafting and Reconstruction of the Middle Ear Sound-Conduction Mechanism*

FREDERICK R. GUILFORD, M.D.; WILLIAM K. WRIGHT, M.D., and W. LEONARD DRAPER, M.D.,
Houston, Texas

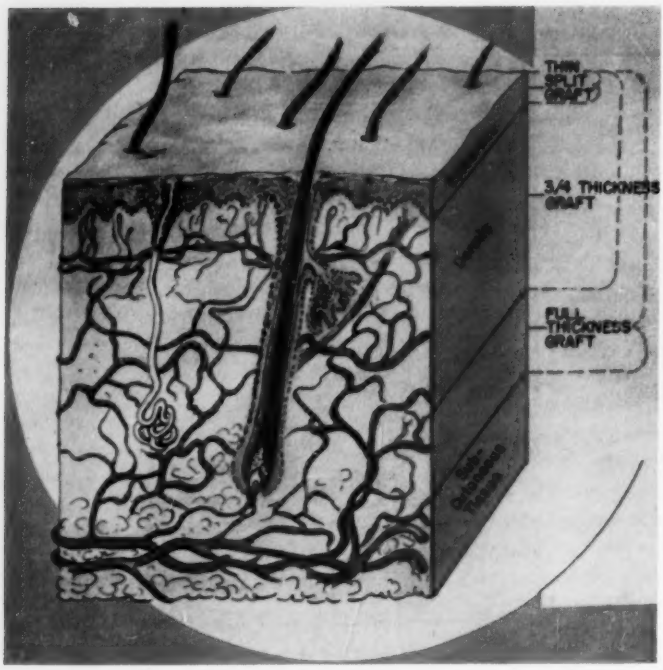


Fig. 1.—Vascular supply of skin, demonstrating necessity of three-fourth-thickness to full-thickness skin graft in order to obtain adequate vascular supply for tympanic graft.

Physiology of Tympanic Skin Grafting

I. A tympanic skin graft stretches across the middle ear. Its bed lies peripherally and consists of the denuded rim of drum, annulus and adjacent canal wall. It flourishes after endothelial vascular buds grow from the bed out into the graft and make continuity with the graft blood vessels to form its permanent blood supply. This occurs about the 4th to 10th day.

Submitted for publication March 3, 1958.

* Illustrated article, based on a Scientific Exhibit at the 62d Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Chicago, October, 1957.

II. A tympanic skin graft survives the first four days because of

A. A plasmic circulation diffusing extracellularly into the graft. This can nourish only the part of the graft directly over the bed.

B. An immediate intravascular circulation of blood from some of the myriad severed graft capillaries which come in contact with some of the myriad severed bed capillaries. This is the only manner by which nourishment can reach the the central portion of the tympanic skin graft which overhangs the

perforation and is not in contact with the bed.

Conclusions

A. Only gentle pressure should be used in packing a tympanic skin graft. (Pressure greater than 10 cm. H_2O pressure might collapse the capillaries and cause slough of the central portion of the graft. We have noted this repeatedly.)

B. Grafts should be cut thick enough to include the network of anastomotic blood vessels running parallel to the skin surface. When they are not included, as in thin split-thickness grafts, central sloughs are common. (We believe that grafts from three-fourths to full thickness make the best tympanic skin grafts. They contain both the superficial and the deep parallel blood supply, which increases the collateral circulation.)

Factors Important in Successful Tympanic Skin Grafting

I. Absence of Infection.
   A. No latent infection (patient should have a dry ear at least 30 days before surgery).
   B. Pre- and postoperative antibiotics (Mysteclin [combination of tetracycline and nystatin] 250 mg., t. i. d., 24 hours preoperatively and 7-10 days postoperatively).
   C. Sterile surgical prep and operating-room technique.
   D. Sterile postoperative technique in office dressings.

II. *Good exposure of the entire tympanic remnant and annulus is crucial.* Endaural incisions and enlargement of the bony canal may be necessary. Exposure at the beginning of surgery is never as good as it seems; and inadequate exposure is a major cause of failure, because
   A. The graft bed must be *completely denuded of every vestige of squamous epithelium* with as little trauma as possible. This demands a binocular magnified view of the entire drum annulus.
   B. The graft must be placed in close approximation with the bed. There should also be an adequate overlap. (This also demands a binocular magnified view of all surfaces.)

III. The skin graft used should be
   A. Thick enough (three-fourths to full thickness) to include the subepithelial anastomotic vascular network.
   B. Atraumatically prepared as follows: 1. Use very sharp blades. 2. Avoid drying by cutting graft just before it is to be used (do not place graft in isotonic saline, which is injurious—if it can't be used immediately, replace it on the donor site). 3. Handle gently—no crushing with forceps or scissors.
   C. A thick split-thickness graft. We feel that this type graft will take more readily than a full-thickness graft.

IV. Almost no pressure should be used in packing graft in position.

V. Regrafting of primary failures within two to three months is more likely to succeed than the original attempt, because the presence of the first graft greatly stimulates the vascularity of the graft bed.

Results of 154 Tympanic Skin Grafts

(Three Months or More Follow-Up)

Complete Closure of Perforation
97 of 154.....63%

Old and Recent Results

(154 Tympanic Skin Grafts)

First 10 Cases	1 Closure	(10%)
Second 10 Cases	4 Closures	(40%)
Last 118 Cases (using present technique)	83 Closures	(71%)
Regrafting of Failures (present technique)	28 Regrafts..23 Closures	(83%)

Conclusions:

1. The present technique, employing a thick skin graft, asepsis, good exposure, and the nonpressure pack, has improved results considerably.

2. Regrafting after a failure has a greater chance of success than the primary attempt.

Follow-Up on Successful Tympanic Skin Graft Closures

Length of Follow-Up	No. of Cases
3-6 Months	32
6-12 Months	32
1-2 Years	14
2-3 Years	14
3-4 Years	5
Total	97

Follow-Up Failures

(154 Tympanic Skin Grafts)

Noted 1-2 Months	49
Noted 2-8 Months	8
Total	57

Conclusion: Tympanic skin grafts once taken tend to endure.

Preliminary Procedures

I. Suppuration must be controlled before tympanic skin grafting is done.

A. Culture and sensitivity tests are obtained, and the patient is started on local and systemic antibiotics.

B. To obtain better contact between drug and infection, secretions are aspirated frequently, and polyps are removed. If a focus in the mastoid fails to resolve, a thorough simple mastoidectomy is done.

C. To create a patent Eustachian tube and prevent recurrent infection, an adenoidectomy and sinus or allergy treatment may be necessary.

D. Except when cholesteatoma is present, these methods will usually produce a dry ear, in which tympanic skin grafting is more likely to succeed. If *radical mastoid surgery should be necessary, however,* it *should* be as conservative as possible, especially as regards the saving of the annulus, and keeping the *Eustachian tube* open. There is little to be gained from sacrificing these structures, and they are very valuable in surgical reconstruction of the middle ear and sound-conduction mechanism by tympanic skin grafting.

Technique Used in Tympanic Skin Grafting

I. Sterile operating room technique.

II. Anesthesia: *2%* lidocaine (Xylocaine) with 1:50,000 epinephrine (Adrenalin) by infiltration; *2%* lidocaine with 1:4,000 epinephrine at external bony canal orifice for hemostasis.

III. Procedure

A. Surgical exposure to binocular magnified vision (loupe or microscope) of the entire circumference of the annulus by

1. U-shaped incision in the skin of the anterior canal wall beginning 1-2 mm. from the drum. A pedicle flap is rolled up onto the cartilaginous canal, and the anterior bony canal overhang is thinned with a sharp curette. The canal skin is rolled back into place at the end of the operation. If this exposure is insufficient,

2. A modified endaural incision may be necessary. The incision is carried down to a point 2 mm. from the drum, and the skin of the posterior canal wall is rolled up as a flap to be replaced at the end of the operation. A self-retaining retractor can then be inserted, which leaves both hands free for instrumentation.

3. Occasionally adequate exposure can be obtained only by enlarging the canal posteriorly with a burr.

B. Removal of the outer layer of squamou epithelium from the rim of the drum and adjacent canal wall to create a graft bed. This is done with tympanic membrane curettes and a Hartmann cup forceps.

C. A thick split-thickness graft is cut with a "Weck" *razor blade* from the non-hair-bearing skin over the mastoid. It is trimmed to overlap the perforation 2-3 mm. in all directions save posteriorly, where it overlaps 5-6 mm.

D. The graft is placed in the ear canal near the drum. It is flattened, and the edges are everted with small sharpe *picks.* The graft is then pushed into contact with the drum and annular groove with a wire *ring curette.*

E. Cigarette paper strips and then 1/16 in. cotton balls are used to gently hold the graft against the annulus. Anterior and posterior wall skin flaps are rolled back

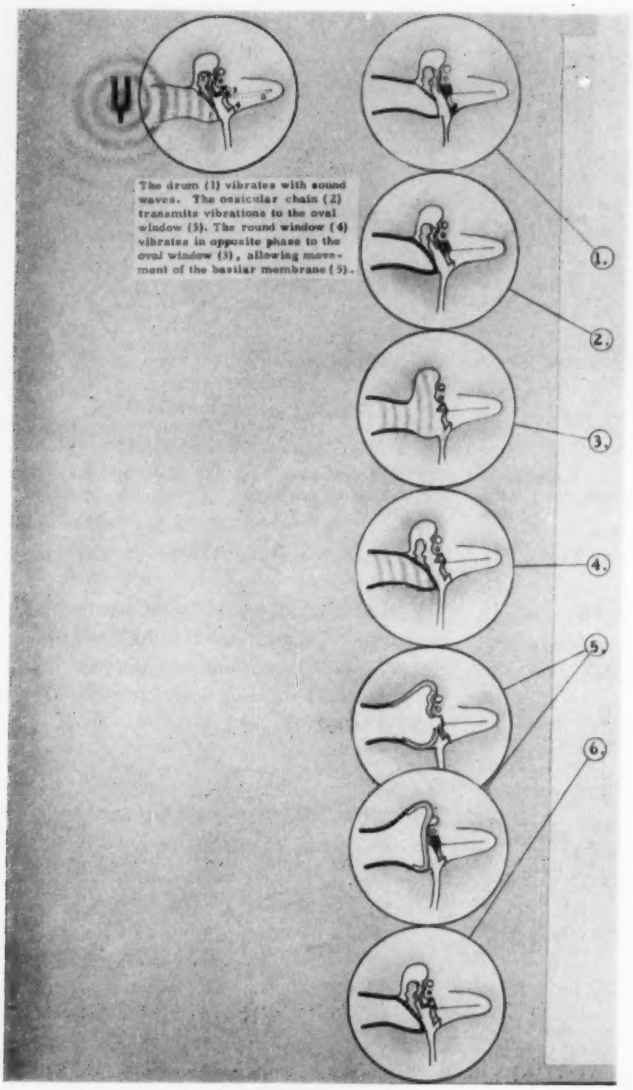

The drum (1) vibrates with sound waves. The ossicular chain (2) transmits vibrations to the oval window (3). The round window (4) vibrates in opposite phase to the oval window (3), allowing movement of the basilar membrane (5).
1.
2.
3.
4.
5.
6.

Figure 2

in position; bare areas are covered with split grafts, and the canal is filled with chlortetracycline (Aureomycin) ointment impregnated cotton balls. The donor site is covered with rayon patch and an elastic Band-Aid. If an endaural incision is made, a mastoid type dressing is applied for two to three days.

IV. Postoperative Management: The patient is kept on Mysteclin 10 days. The packs are removed on the 7th day and the cigarette paper at 14 days. The donor site is healed in 10 days.

Physiology of Middle-Ear Sound Conduction

The basis of cochlear function is to retain or create two freely movable windows (one in contact with each scala of the cochlea).†

1. If there are no functional openings, total deafness occurs for both air conduc-

† Numbers on illustration correspond to paragraph numbers.

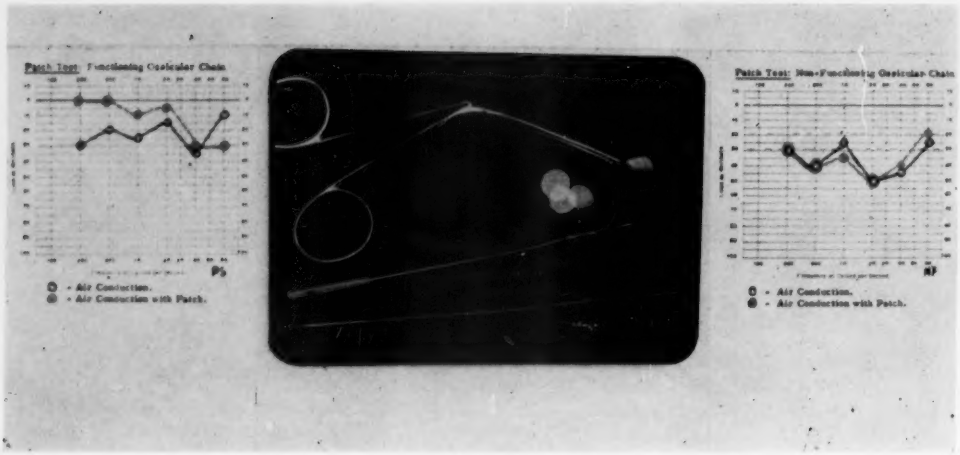


Fig. 3.—Patch test. Illustrative audiograms showing functioning ossicular chain and nonfunctioning ossicular chain before and after patching in each audiogram.

tion and bone conduction, from immobility of the labyrinthine fluids.

2. If only one window is open, as in typical otosclerosis, there is usually about a 60 db. air-conduction loss, and bone conduction is normal except for a slight depression (the otosclerotic notch), resulting from partial immobilization of the labyrinthine fluids.

3. If two windows are open, but both are exposed to direct sound vibration, as in total absence of the tympanic membrane, there is some cancellation of sound impulse from a wave striking both windows at almost

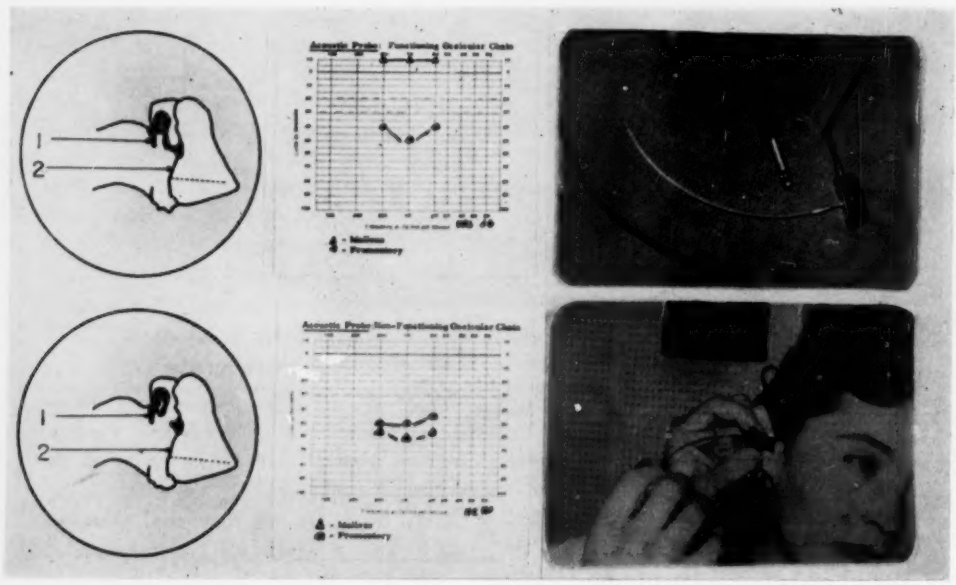


Fig. 4.—Acoustic probe test. Showing functioning ossicular chain and nonfunctioning ossicular chain results when acoustic probe is used.

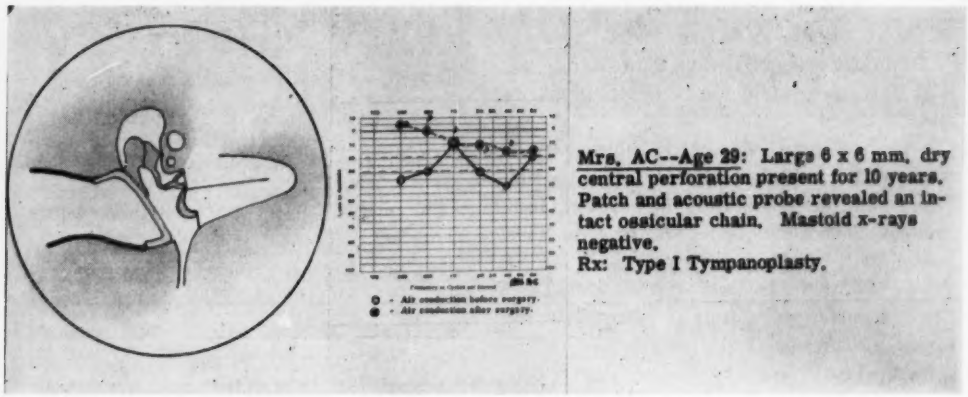

Mrs. AC--Age 29: Large 6 x 6 mm. dry central perforation present for 10 years. Patch and acoustic probe revealed an intact ossicular chain. Mastoid x-rays negative.
Rx: Type I Tympanoplasty.

Fig. 5.—Tympanic skin grafting Type I. Showing hearing level before and after surgery. Average preoperative level for speech frequencies: 23 db. Average postoperative level for speech frequencies: 7 db.

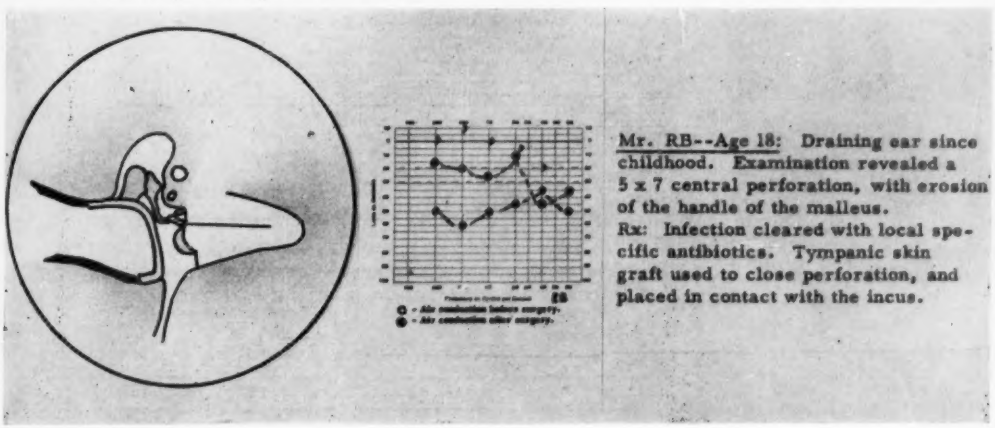
the same instant. The hearing loss for air conduction is at about the 50 db. level. Bone conduction is normal.

4. Protection of both windows from direct sound, which occurs in discontinuity of the ossicular chain in the presence of an intact ear drum usually produces about a 60 db. loss.

5. If one window (the round window) can be protected from direct sound vibration while the other is directly exposed to sound, the hearing loss is only 20 db. This is the situation after a successful fenestration and a Type IV or V tympanoplasty.

6. When direct sound strikes an intact ear drum and is transmitted to the oval window via the ossicular chain, while at the same time the round window is protected from sound, the final 20 db. of conduction deafness is restored. Most of this last 20 db. of gain is from the mechanical advantage of the larger vibrating ear drum being transmitted to the smaller oval window. The ossicular chain has an amplifying power equivalent to only 1.7 db., and its chief value is that of sound transmission.

Fig. 6.—Tympanic skin grafting Type IIA. Showing hearing level before and after surgery. Average preoperative level for speech frequencies: 52 db. Average postoperative level for speech frequencies: 18 db.


Mr. RB--Age 18: Draining ear since childhood. Examination revealed a 5 x 7 central perforation, with erosion of the handle of the malleus.
Rx: Infection cleared with local specific antibiotics. Tympanic skin graft used to close perforation, and placed in contact with the incus.

Special Audiometric Tests

Mere closure of a perforated tympanic membrane will not always improve hearing. In fact, in certain cases hearing will be made worse.

With a knowledge of the physiology of middle ear sound conduction, it is possible to adapt the tympanic skin grafting technique to achieve better hearing in most situations. Preoperative evaluation of the integrity of the ossicular chain by the patch and acoustic probe tests is essential in this type of surgery.

Patch Test

If closing the perforation with a cigarette paper patch increases hearing to near the bone conduction level, the ossicular chain is presumably functional.

Acoustic Probe Test

The acoustic probe is a point bone conductor which can be attached to an audiometer. Hearing tests are made with the probe in contact with (1) the malleus; (2) the promontory.

Modifications of Tympanic Skin Grafting

Type I

Pathology: Simple perforated tympanic membrane with intact ossicular chain.

Rx: Skin graft closure of the perforation will result in essentially normal hearing.

Type IIA

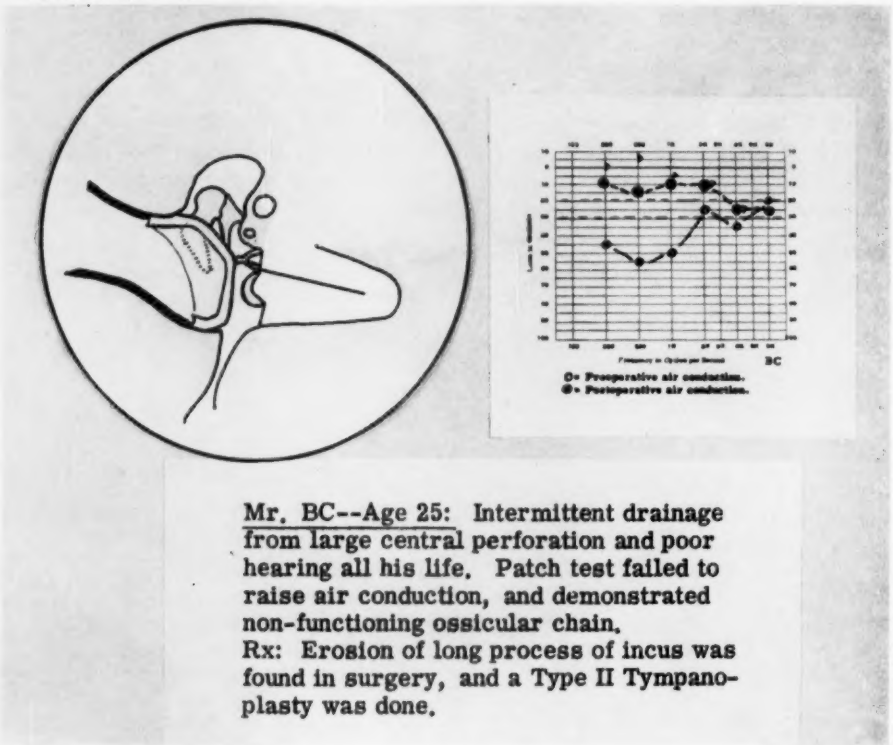
Pathology: Perforated tympanic membrane with erosion of malleus.

Rx: The graft is placed so that it contacts the incus.

Type IIB

Pathology: Necrosis of the end of the long bone process of the incus ("anemic

Fig. 7.—Tympanic skin grafting Type II. Showing hearing level before and after surgery. Average preoperative level for speech frequencies: 43 db. Average postoperative level for speech frequencies: 12 db.


BC
O• Preoperative air conduction.
●• Postoperative air conduction.
Mr. BC--Age 25: Intermittent drainage from large central perforation and poor hearing all his life. Patch test failed to raise air conduction, and demonstrated non-functioning ossicular chain.
Rx: Erosion of long process of incus was found in surgery, and a Type II Tympanoplasty was done.

necrosis"—Tumarkin) with disruption of the incudostapedial joint is not uncommon following chronic middle-ear suppuration. (Fibrous adhesions which may bridge the gap between the incus and the head of the stapes do not act as a satisfactory incudostapedial joint in transmitting sound impulses.)

Rx: In the presence of disruption of the incudostapedial joint, the accepted procedure has been to remove the ossicles in conjunction with radical mastoidectomy and the Wullstein Type III tympanoplasty. We have found that this type of pathology can often be handled by modifications of Wullstein's Type II tympanoplasty.

This technique has the following advantages:

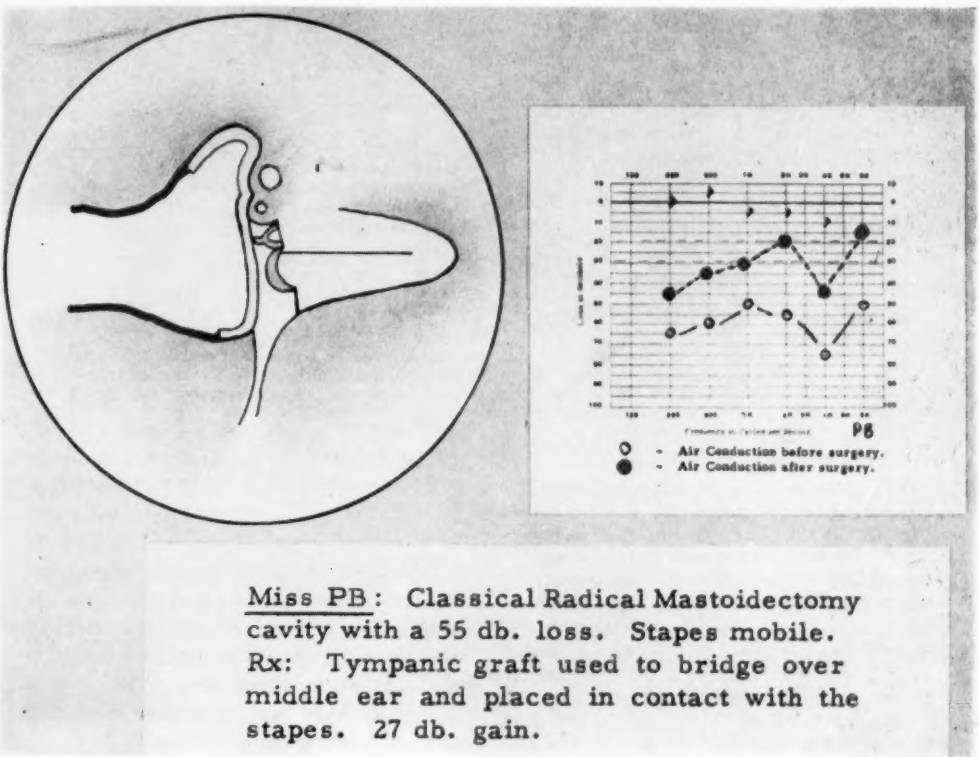
A. It can often be done directly through the external auditory canal, as in a Type I tympanoplasty (myringoplasty). (If the status of the mastoid is unsure, exploration of the mastoid, including the epitympanum may be done, leaving the bony external auditory canal intact. If the epitympanum is free of disease, it is often unnecessary to remove the incus.)

B. It insures a good air space in the stapes area of the middle ear, allowing free mobility of the new tympanic membrane and stapes.

C. Improvement of hearing may be greater than the average Type III tympanoplasty result.

D. It avoids the inherent disadvantages of a radical mastoidectomy cavity. (However, this technique may be applied when a modified mastoidectomy is necessary, *if a small portion of the bridge is left intact*.)

Fig. 8.—Tympanic skin grafting Type III. Showing hearing level before and after surgery. Average preoperative level for speech frequencies: 55 db. Average postoperative level for speech frequencies: 28 db.


PB
- Air Conduction before surgery.
- Air Conduction after surgery.
Miss PB: Classical Radical Mastoidectomy cavity with a 55 db. loss. Stapes mobile.
Rx: Tympanic graft used to bridge over middle ear and placed in contact with the stapes. 27 db. gain.

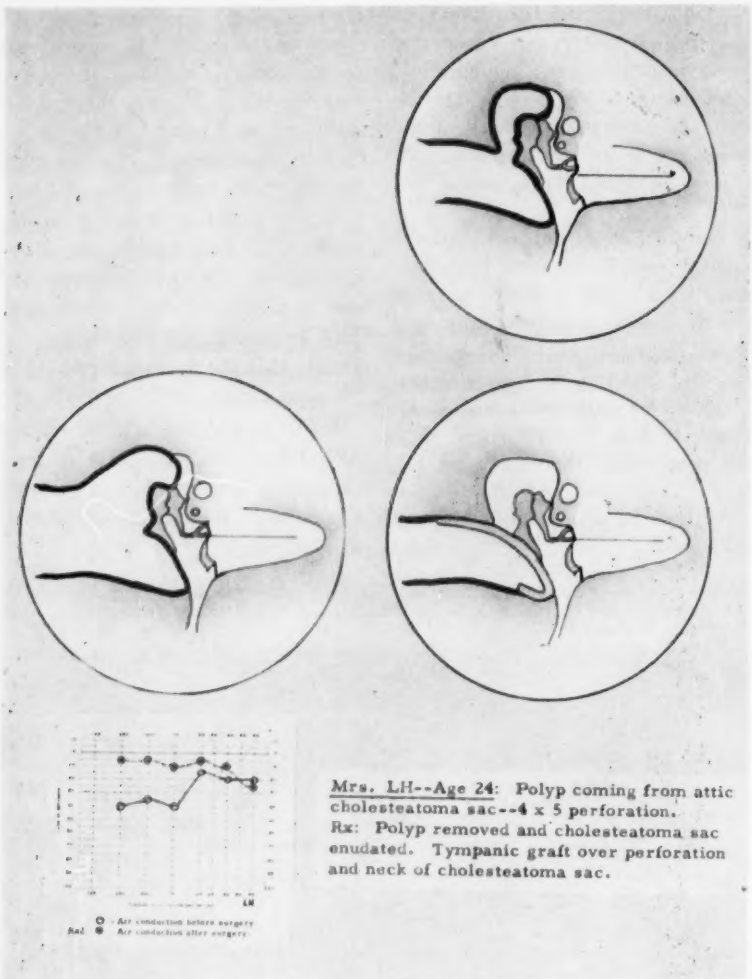

Mrs. LH--Age 24: Polyp coming from attic cholesteatoma sac--4 x 5 perforation.
Rx: Polyp removed and cholesteatoma sac enudated. Tympanic graft over perforation and neck of cholesteatoma sac.
LH
Air conduction before surgery
Air conduction after surgery

Fig. 9.—Above, small cholesteatoma, exteriorized by modified radical mastoidectomy and by removal of cholesteatoma sac and closure of perforation by skin graft. Below, illustrative audiogram showing hearing before and after surgery. Average preoperative level for speech frequencies: 30 db. Average postoperative level for speech frequencies: 7 db.

Technique:

A. The denuded tympanic remnant is raised from the annular sulcus posteriorly, and the incudostapedial joint area is exposed by wide removal of bone in the annular area (wider removal of bone than is required in stapes mobilization). At this time the round window area may be inspected and mobility of the stapes determined.

B. When the graft is applied, the denuded remnant of drum and graft are depressed and packed lightly in place so as to contact the shortened long process of the incus, as well as the head of the stapes. Thus the graft bridges from the annulus anteriorly to the malleus, to the incus, to the stapes, and back on to the posterior wall of the canal.

Type III

Pathology: Loss of ossicular continuity, but the stapes is mobile and the crura are intact. Loss of posterior and superior canal wall because a radical mastoidectomy is necessary or has been done. The annulus and Eustachian tube have been preserved and are functional.

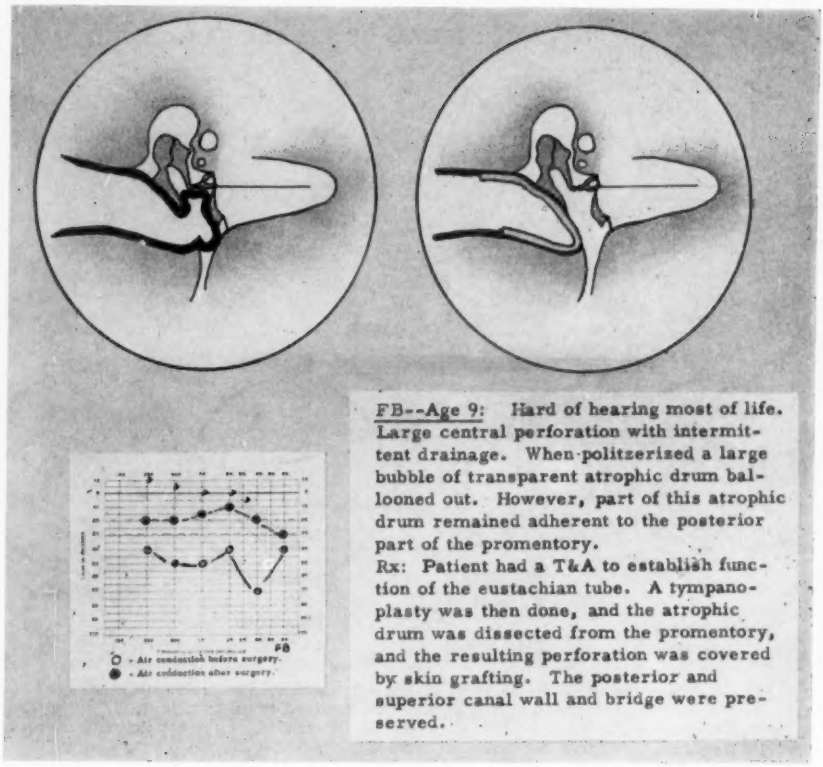

FB--Age 9: Hard of hearing most of life. Large central perforation with intermittent drainage. When politzerized a large bubble of transparent atrophic drum ballooned out. However, part of this atrophic drum remained adherent to the posterior part of the promontory.
Rx: Patient had a T&A to establish function of the eustachian tube. A tympanoplasty was then done, and the atrophic drum was dissected from the promontory, and the resulting perforation was covered by skin grafting. The posterior and superior canal wall and bridge were preserved.
FB
○ - Air conduction before surgery.
● - Air conduction after surgery.

Fig. 10.—Above, retraction pocket and repair of perforation after pocket is removed. Below, illustrative audiogram, pre- and postoperative. Average preoperative level for speech frequencies: 47 db. Average postoperative level for speech frequencies: 15 db.

Rx: The graft bridges from the annulus to the floor of the attic and facial ridge but contacts the stapes.

Complications

Routine use of tympanic skin grafting to close the middle ear space can, under certain conditions, be harmful. These conditions must routinely be tested for preoperatively, so that they may be avoided.

I. Cholesteatoma.
   A. Diagnostic Measures.
      1. Mastoid x-rays.
      2. Presence of cholesteatomatous material.
      3. Presence of marginal or attic perforations.
      4. *Inspection of the middle ear under magnification for the presence of squamous epithelium.*

   (If there is any suspicion of cholesteatoma an exploration of the mastoid and atticus should be done.)
   B. Treatment.
      1. The cholesteatoma sac can be exteriorized by a modified radical mastoidectomy.
      2. The cholesteatoma sac can sometimes be completely removed, and the mouth of the sac closed by skin grafting.

II. Retraction Pocket. What appears to be a perforation may be an atrophic area of tympanic membrane sucked inside and often more or less adherent to the walls of the middle ear.

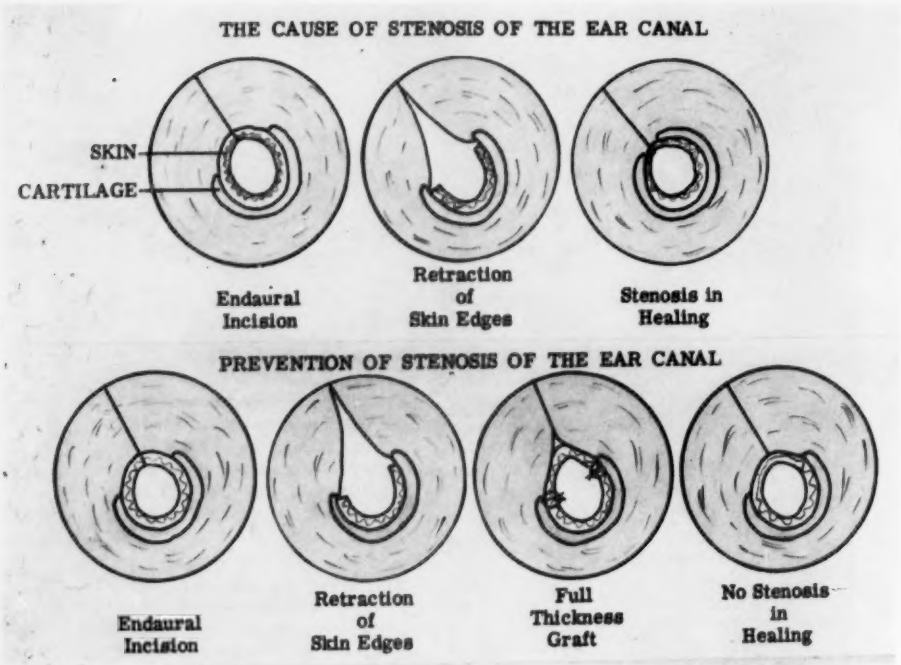


Fig. 11.—Above, the cause of stenosis of the ear canal. After an endaural incision the skin edges retract beyond the margins of the ear cartilage, thus tending to cause stenosis during the healing of the incision. Below, prevention of stenosis of the ear canal by suturing a full-thickness graft between the retracted skin edges.

A. Diagnostic Measures.
   1. Politzerization—A bubble of transparent drum will balloon out of the perforation.
   2. Pneumatic otoscope.
   3. Inspection of the middle-ear promontory under magnification for squamous epithelium.

B. Treatment.
   1. The Eustachian tube must be made functional by Rx of adenoids, sinuses, allergy, etc., then—
   2. The middle ear is explored, and the atrophic drum is dissected from the lining of the middle ear. The resulting perforation is closed by skin grafting.

III. Nonfunctioning Eustachian Tube. If the Eustachian tube does not ventilate the middle ear, skin graft closure of a perforation will cause an annoying middle-ear effusion, and often increase the hearing loss.

A. Diagnostic Measures.
   1. Politzerization.
   2. Calibrated Valsalva test.
   3. Hearing of a nasal sound source at the ear when the patient swallows.

B. Treatment.
   1. Rx of adenoids, sinuses, allergy, etc.
   2. If the Eustachian tube cannot be successfully restored to function, the perforation should not be closed.

IV. Hearing Made Worse. Closure of a perforation in the presence of a nonfunctioning ossicular chain or a nonfunctioning Eustachian tube may make the hearing worse. It is essential to predetermine these conditions by the patch and acoustic probe tests, and

Eustachian tube "function tests," and to modify the surgical technique accordingly, as explained above.

V. Draining Ear. Sterile operating room and postoperative technique and the use of prophylactic antibiotics will usually prevent this complication. If infection should develop, culture and specific local and systemic antibiotics are indicated.

VI. Stenosis of the Ear Canal. Any endaural incision tends to heal with a slight stenosis. In abnormally small canals postoperative stenosis can be avoided by suturing a wedge-shaped, 1 cm.-wide skin graft between the edges of the incision in the cartilaginous canal at the end of the operation.

6410 Fannin (25).

Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Modifications of Cochlear Responses by Oxygen Deprivation

CÉSAR FERNÁNDEZ, M.D., and RENÉ ALZATE, M.D., Chicago

Several workers have investigated the susceptibility of cochlear potentials to oxygen deprivation. The results showed that cochlear microphonics, action potentials, and DC resting potential are oxygen-dependent. Békésy [1] demonstrated that both cochlear microphonics and DC resting potential, as recorded from scala media, followed the same time-course decay during anoxia. Wever et al.[2] reported that in the cat the amplitude of cochlear microphonics diminished when the concentration of oxygen in the respirating gas mixture was below 4%. Lawrence and Wever [3] reported a detectable lesion in the organ of Corti of cats exposed to severe oxygen deprivation. However, Falbe-Hansen et al.[4] did not find such changes in guinea pigs and cats exposed to either acute or prolonged oxygen lack. Bornschein and Gernandt [5] showed that by repeating the exposure to anoxia, the neural components can be removed selectively from cochlear microphonics. But according to other investigations [6] this method seemed to induce large depression of cochlear microphonics also.

The investigation to be described here was undertaken with the following purposes: (1) to compare the behavior of cochlear microphonics and action potentials during and after several types of oxygen deprivation; (2) to induce permanent depression of cochlear responses by following the method of Bornschein and Gernandt,[5] so that acute and chronic preparations, suitable for histologic studies, might be obtained; (3) to determine the survival of anesthetized and unanesthetized guinea pigs to different conditions of oxygen deprivation.

Submitted for publication May 20, 1958.

From the Division of Otolaryngology of the University of Chicago.

Partially supported by Research Grant B-682 (C3) from the National Institute of Health and by Contract AF 41 (657) 143 with the School of Aviation Medicine, U. S. A. F., Randolph Field Air Force Base, Texas.

Methods

This investigation was done using guinea pigs of about 300 gm. Anesthesia—when used—was with diallylbarbituric acid (Dial) in urethan (ethyl carbamate) 0.50 cc. per kilogram body weight.

The procedure for implanting a pair of electrodes in the first turn of the cochlea, as described elsewhere,[7] was used. The cochlear responses after proper amplification were measured or photographed from a dual-beam cathode-ray oscillograph before, during, and after each bout of oxygen deprivation. The heart rate was followed by means of a loud-speaker coupled to electrodes implanted in the hind leg and neck.

The stimuli were 7,000, 8,000 or 10,000 cps tone pips which were obtained by passing a pure sinusoidal wave through an electronic gate designed and constructed by Dr. J. Hind. The transducer was coupled with a plastic tube (85 mm. in length) to a speculum sewed in the animal's ear. The intensity of acoustic stimulus was generally kept constant at a level just below nonlinearity of cochlear mircophonics.

To investigate the behavior of cochlear responses, oxygen deprivation was carried out in 25 anesthetized guinea pigs. The animals were maintained continuously under artificial respiration, and oxygen deprivation was induced in the following ways: (1) hypoxia, where a gas mixture of nitrogen with

5%, 2.4%, or 1% oxygen was used; (2) fulminating anoxia indicating respiration of pure nitrogen, and (3) asphyxia, in which case the trachea was clamped.

Hypoxia and fulminating anoxia were induced by connecting the respirator to tanks containing known gas mixtures. The duration of each bout of oxygen deprivation was 2 minutes, after which the respirator was connected again to normal air. The bout was repeated at one-hour intervals, following the order mentioned above (hypoxia, fulminating anoxia, asphyxia) or the order was reversed (asphyxia, fulminating anoxia, hypoxia) or the bouts were intermingled. Some animals received only one bout of oxygen deprivation which was prolonged until the animal died.

In order to induce permanent depression of cochlear responses, the method of Bornschein and Gernandt [5] was followed in 12 guinea pigs curarized with purified chondodendron tomentosum extract (Intocostrin) (0.10 cc. intramuscular in single dose). Five of these animals were exposed to bouts of asphyxia (3-7 minutes' duration each), which were repeated at one-hour intervals until the responses were depressed. The other seven were exposed only once to asphyxia (5-6 minutes' duration) which was not repeated because during recovery heart irregularities appeared. These 12 guinea pigs were killed at the end of the experiment by intravital fixation with Heidenhain-Susa. The temporal bones were removed in one block and treated by the routine method for hematoxylin-eosin stain. The histological preparations were studied with regular light microscopy. This group of animals we shall refer to as acute experiments.

Another group of five unanesthetized guinea pigs were exposed to prolonged oxygen deprivation. The animals were enclosed in a small plastic chamber through which a gas mixture of nitrogen with about 3% oxygen, flowed at a rate of 4 liters per minute. Two of them remained in this gas mixture atmosphere for about 30-45 minutes. The exposure was repeated three times the same day and again one and four days later. They were kept alive for 40 days; the hearing was tested by the usual electrophysiological method; the animal killed by intravital fixation, and the temporal bones saved for histological studies. The other three guinea pigs were exposed only once to the same gas mixture (3% oxygen) for 2 consecutive hours. One at a time was killed 7, 14, and 17 days later by intravital fixation. The hearing was tested in the usual manner. This group we shall refer to as chronic experiments.

To investigate the effect of anesthesia on the survival of the animal as a whole, a group of 35 guinea pigs, with and without anesthesia were enclosed in a small plastic chamber through which several known gas mixtures were blown at the rate of 4 liters per minute. Other anesthetized guinea pigs were exposed to the same gas mixtures, but this was given by means of a respirator. Samples of the electrocardiogram taken at short regular intervals allowed identification of the moment the heart stopped beating. Some animals survived a long time, in which case the experiment was discontinued after 5 hours. In this group neither measurements of cochlear responses nor histologic studies of temporal bones were made.

Blood pressure, pO_2 and pCO_2 measurements were not determined in this investigation.

Survival of Guinea Pigs Under Different Conditions of Oxygen Deprivation

	Survival Time, Minutes		
	Unanesthetized	Anesthetized	Respirator *
Asphyxia	11-24	22	
Fulminating anoxia	9-13	17-29	14-17
Hypoxia (1% O_2)	9-15	9-28	50
Hypoxia (2.4% O_2)	18-22	24	250
Hypoxia (5% O_2)	>340	66	>300

* Respirator was used on anesthetized animals.

Results

A. Survival of Guinea Pigs During Oxygen Deprivation.—The Table shows the survival of guinea pigs during different conditions of oxygen deprivation. Unanesthetized guinea pigs survived in an atmosphere of nitrogen with 5% oxygen until the experiment was discontinued after 5 hours. Under anesthesia the survival was about 70 minutes. Under the other con-

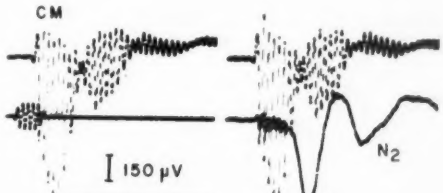


Fig. 1.—Cochlear responses to a 10,000 cps tone pip at high intensity as recorded with differential electrodes placed at about 4 mm. from the round window. The record on the left shows cochlear microphonics (CM) and the acoustic signal. The record on the right illustrates CM and neural components (N_1 and N_2). The photographs were taken from the screen of a Tektronix 532 cathode-ray oscilloscope with a dual-trace unit (Type 53/54 C). Time calibration, 10,000 cps.

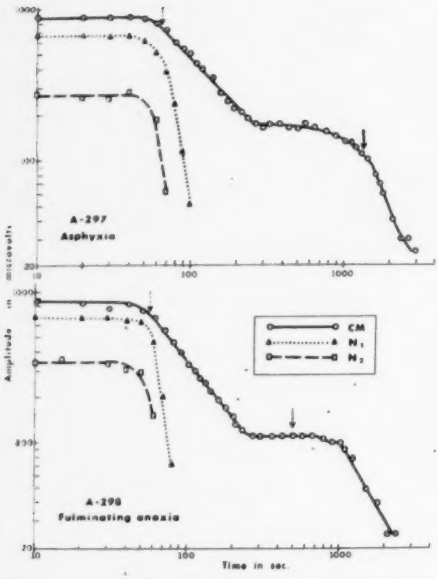


Fig. 2.—Survival of cochlear responses during asphyxia and fulminating anoxia. The broken line arrow indicates the point where heart irregularities appeared, and solid line arrow when heart stopped. Notice that both time and amplitude of responses are plotted in logarithmic scale.

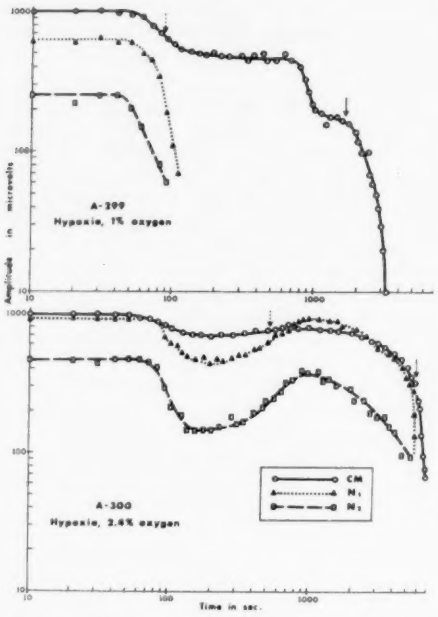


Fig. 3.—Survival of cochlear responses during hypoxia 1% and 2.4% oxygen. The broken line arrow indicates the point where heart irregularities appeared, and solid line arrow when heart stopped. Logarithmic scale.

ditions, with or without anesthesia, the heart stopped in 12 to 25 minutes. Individual variations in survival were observed mainly among the unanesthetized guinea pigs.

The respirator modified considerably the survival of the anesthetized animals. When the gas mixture contained 1% oxygen, the survival increased two to three times; with 2.4% oxygen the increase was 10 times, and with 5% oxygen the animal survived a period of 5 hours.

B. Latency of Cochlear Responses.—The symbols and nomenclature which identified the different potentials are those used by Davis.[8] CM_1 indicates the oxygen-dependent part of cochlear microphonics; CM_2 the anaerobic part, and CM an unspecified mixture of both. N_1 indicates the first action potential which appears after CM is initiated and N_2 the action potential which appears after N_1. Figure 1 illustrates a typical response as obtained by differential electrodes from the first turn of the cochlea. The endolymphatic potential (DC resting potential of Békésy[9]) is symbolized by EP and the intracellular potential of hair cells by DC.

Latency of CM is defined as the time interval from beginning of the stimulus to the foot of the first wave of cochlear microphonics. In normal guinea pigs the latency of CM was about 0.35 msec. and remained constant under any circumstance of oxygen deprivation.

Latency of N_1 and N_2 was defined as the time interval between the beginning of CM and the negative peaks of N_1 and N_2 respectively. For 10,000 cps tone pips kept at constant intensity (about 55 db. above threshold for N_1) the latency of N_1 and N_2 increased during oxygen lack by as much as 0.3 msec.

C. Survival of Cochlear Responses.—Survival of CM, N_1, or N_2 was defined as the time interval between the beginning of

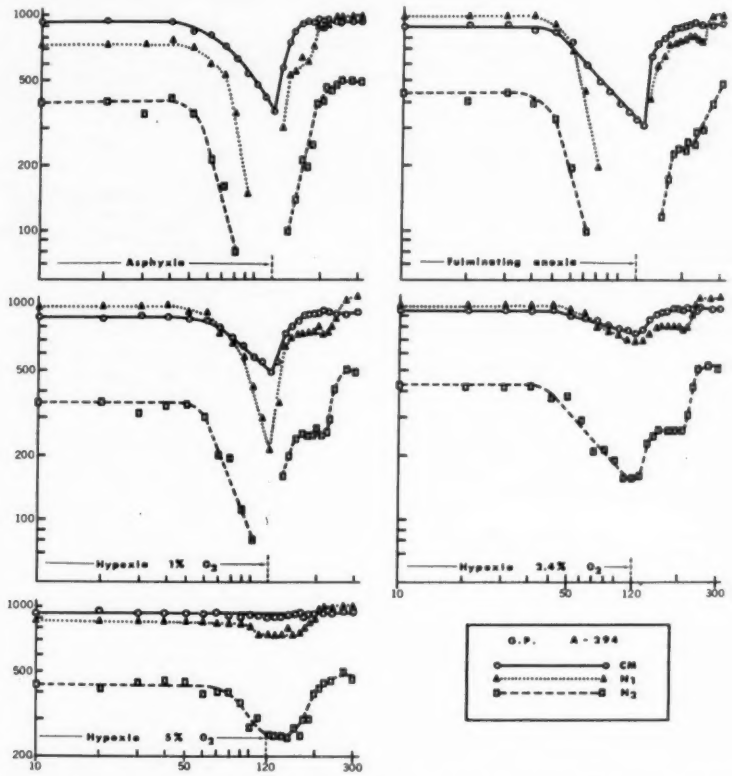


Fig. 4.—Changes in amplitude of cochlear responses during 2 minutes of oxygen deprivation and recovery after air was again available. Cochlear microphonics are not only more resistant to oxygen deprivation than neural components but also showed a remarkably fast recovery. The exposures were given in the following order: fulminating anoxia, hypoxia 1% O_2, hypoxia 2.4% O_2, hypoxia 5% O_2, and finally asphyxia. Notice that CM consistently recovered its original amplitude while N_1 shows an accumulative depression from one exposure to the next; the initial voltage of N_1 was 1,000 μV and final voltage was 750 μV. N_2 also shows some degree of accumulative depression. Abscissa, time in seconds (logarithmic scale) and ordinate, amplitude of cochlear microphonics in microvolts (logarithmic scale).

any bout and the moment the responses disappeared.

In Figures 2 and 3 the amplitude of CM, N_1, and N_2 during different conditions are plotted against time. In asphyxia, fulminating anoxia, and hypoxia—1% oxygen—a change in the steady state of the three potentials was consistently observed 50-60 seconds after either condition was initiated. The change to unsteady state preceded or was coincidental with heart irregularities. In hypoxia—2.4% oxygen—the first signs of depression in cochlear responses appeared at about 80 seconds and in hypoxia —5% oxygen—still later, at about 90 seconds. In these two conditions heart disturbances appeared after the potentials changed to an unsteady state.

The decay and survival of cochlear responses varied from one type of oxygen deprivation to another, but asphyxia and fulminating anoxia (Fig. 2) gave approximately the same result. Under these two conditions the decay of N_1 and N_2 was rather fast so that their survivals were about 100 seconds and 60 seconds respectively. In both conditions, the decay of CM was slower than that of neural components.

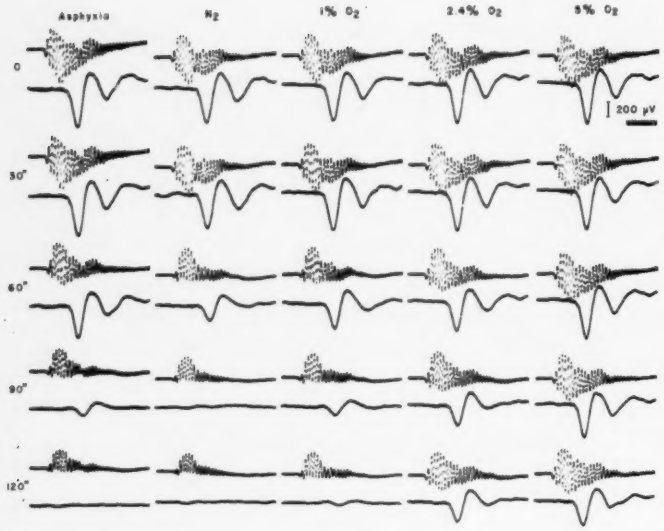


Fig. 5. — Guinea Pig A-293. Changes in cochlear responses during different conditions of oxygen lack. The duration of each bout was 2 minutes. These records were taken every 30 seconds as shown at the left of the figure. The exposures were given at one hour intervals in the order indicated at the top. In this animal the responses decayed faster in fulminating anoxia than in asphyxia, but in general, the decay was about the same in both conditions.

As Figure 2 shows, there was a sudden break in the curve of CM at about 300 seconds. This point marked the boundary between CM_1 and CM_2. The decay of CM_2 was by steps and it survived for hours after death.

In hypoxia with 1% oxygen (Fig. 3) the behavior of neural components was about the same as in the previous conditions, although their survivals were longer (110 seconds for N_1 and 90 seconds for N_2). However, the decay of CM was different. It followed a step pattern in which the boundary between CM_1 and CM_2 could not be identified reliably.

In hypoxia with 2.4% oxygen (Fig. 3) there was an initial drop in the amplitude of responses, but after a few minutes they recovered, sometimes even to their original values. This was followed by a slow decay

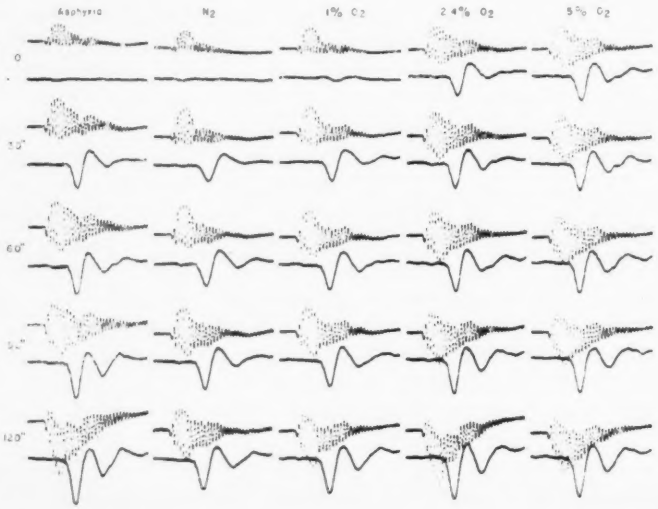


Fig. 6. — Guinea Pig A-293. Recovery of cochlear responses. These records were taken at the times indicated at the left of the figure. Notice that the records shown in row 0 correspond to those in row 120" of Figure 5. Voltage and time calibration are the same as in Figure 5.

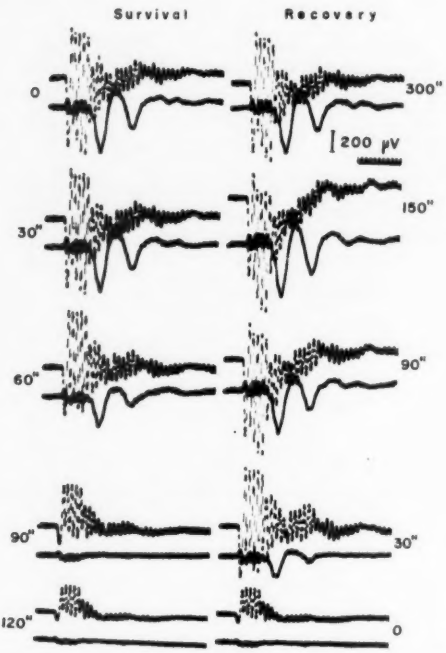


Fig. 7.—Guinea Pig A-295. Overshooting of neural components and summating potential after an exposure of 2 minutes of asphyxia. Time of recovery is counted from the moment air was again available. Notice the downward displacement of the baseline (SP) at 150 seconds, and overshooting of neural components. Time calibration 10,000 cps.

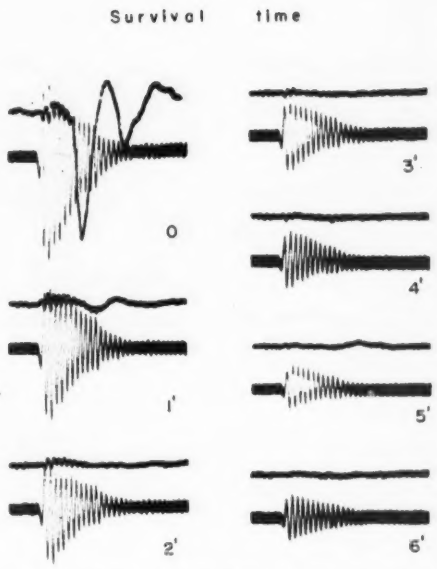


Fig. 8.—Survival time of cochlear responses of Guinea Pig A-37. This animal received four consecutive asphyxias (3, 3, 4, and 6 minutes respectively) with a 1-hour rest period between each. The figure illustrates the survival time of cochlear responses during the fourth asphyxia. The previous exposures to oxygen lack have already produced an accumulative depression of both action potentials and microphonics.

NOTE: In this and subsequent figures, dotted lines are retouched traces made under binocular microscope of traces which can be found in the originals. The peaks are not retouched.

until heart irregularities appeared, at which time the responses deteriorated rapidly.

In hypoxia with 5% oxygen, the responses followed the same time course as in the previous condition but the decay was slower.

D. Recovery of Cochlear Responses.—Recovery was defined as the time interval between the end of a bout and the moment the responses reached their original amplitude.

Figure 4 illustrates the decay of CM, N_1, and N_2, and recovery when air was again available. CM was the first potential to give signs of recovery in about 10 seconds and reached its original amplitude in 70 additional seconds at the most. N_1 lagged behind recovery of CM by a few seconds, but the increase of the spike was generally by steps of fast and slow growth; it reached the preexposure level within 3 minutes. N_2 lagged behind N_1 by as much as 20 seconds, and, like the latter, generally increased by steps. For comparison a case is presented in Figures 5 and 6. Figure 5 shows the records taken during 2 minutes of oxygen deprivation, and Figure 6, those taken during 2 minutes after air was available.

The recovery of neural components was consistently followed by an overshooting period which was coincidental with an increase in the summating potential, as Figure 7 illustrates. This supernormal period lasted for 3 to 4 minutes and in some cases was followed by a period of slight depression of neural components. Occasionally a small overshooting of CM was

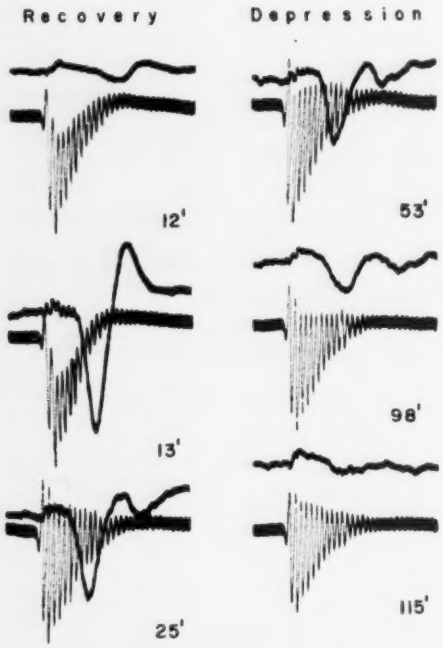


Fig. 9.—Recovery of cochlear responses of Guinea Pig A-37, after the asphyxia illustrated in Figure 8. Time is measured in minutes from beginning of the asphyxic period. The responses start to recover between 5 and 6 minutes after the end of the exposure, which is a significantly longer interval than the recovery time obtained in control animals. After recovery has begun, the responses proceed rapidly through the charactistic overshooting and later, a progressive depression was observed until complete extinction of action potentials at the end of 2 hours. Also the microphonics were significantly reduced in amplitude. During this interval no change in the heart beat was observed. The animal was sacrificed with intravital fixation 10 minutes after the last picture illustrated in the figure was taken. Photomicrographs of the organ of Corti, macula utricularis and one crista ampullaris are presented in Figure 10.

observed, which preceded and faded out before that of N_1 and N_2.

E. Histological Studies.—1. Acute Experiments: The results already described clearly showed that the depression of cochlear responses observed during two minutes of oxygen deprivation was essentially reversible. The bouts can be repeated many times without the responses deteriorating, provided the duration of each bout is no more than 2 minutes, and a 1-hour interval is allowed between exposures. However, N_1 may be partially depressed, as Figure 4 shows (see legend). In order to produce a permanent depression of the responses, the duration of each bout of asphyxia was prolonged to 3-6 minutes, and repeated several times at 1-hour intervals. This effect is illustrated in Figures 8 and 9. The records belong to an animal which was exposed to four consecutive bouts of asphyxia (3, 3, 4, and 6 minutes each). The last one is shown in Figure 8, and the changes in potentials after air was again available are illustrated in Figure 9. Notice in this figure the delayed recovery and subsequent depression, so that at the end of 2 hours, action potentials are absent and amplitude of cochlear microphonics reduced. Ten minutes after the last picture was taken the animal was killed by intravital fixation with Heidenhain-Susa. The study of histologic preparations of temporal bones showed a normal microscopic cytoarchitecture of the inner ear, as Figure 10 illustrates. Five animals prepared in the same manner gave physiologic and histologic results similar to those just described.

Some animals exposed to a long period of asphyxia (4-6 minutes) presented irregularities in the heart rate (arrhythmia, bradycardia) during or after the recovery period. In these cases the cochlear responses were immediately depressed. If the heart recovered in a few minutes, the cochlear responses also recovered, but, if the heart disturbance lasted for a relatively long time (15 minutes or more), an apparently permanent depression was obtained. Such a case is illustrated in Figures 11 and 12. The records belong to an animal exposed to a period of 5 minutes of asphyxia (Fig. 11). The recovery of responses followed the usual pattern (Fig. 12, Recovery) but at about 20 minutes heart irregularities appeared and remained until the experiment was discontinued three hours later. Heart irregularity was followed by a progressive depression of cochlear responses (Fig. 12, Depression) until neural components were barely visible and amplitude of cochlear

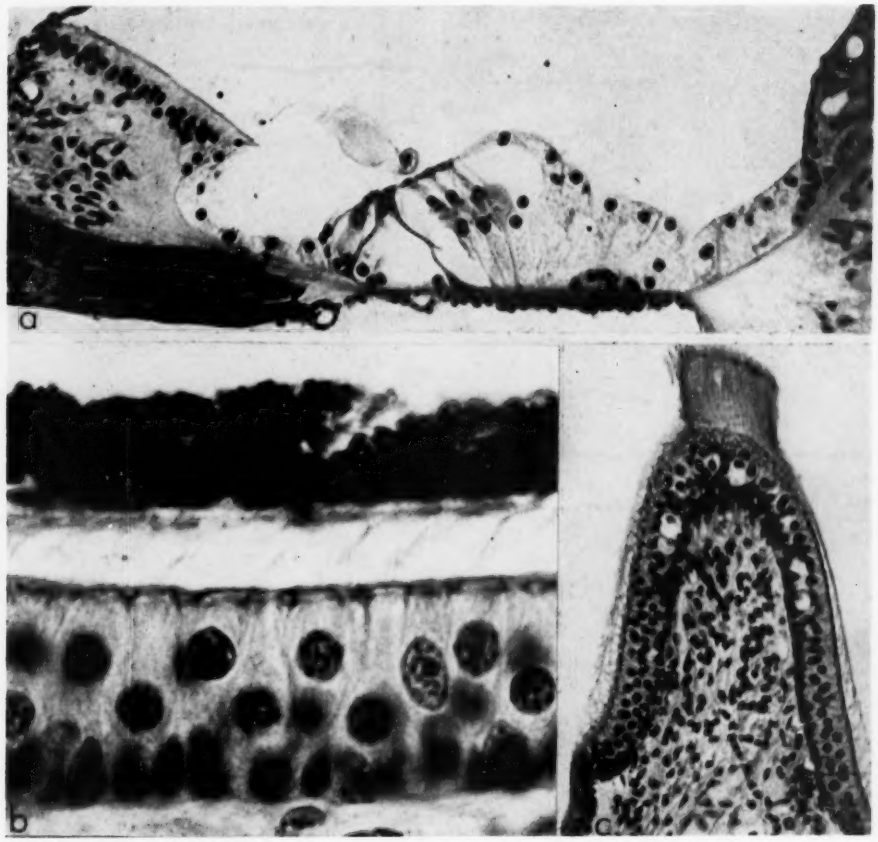


Fig. 10.—Guinea Pig No. A-37. (*a*) Normal organ of Corti (300×). No injury was found in either cochlea. (*b*) Normal structures of macula utricularis (720×). (*c*) Crista of the superior semicircular canal (240×). The arrow points to vacuolation of some sensory cells. Vacuolation of the same sort was found in the other cristae on both sides, but not in the maculae utricularis or sacularis. Whether this vacuolation is an artifact or pathological change induced by impaired metabolism is a question for further investigation. However, only this animal showed such a change in the cells of the cristae, which suggests artifact more than pathology.

microphonics reduced by half. The animal was killed by intravital fixation, and the histological studies of temporal bones showed normal microscopic cytoarchitecture of the inner ear, as Figure 13 illustrates. Six other guinea pigs presented similar heart irregularities and depression of cochlear responses. No changes in the inner ear structures were found in any of these animals.

2. Chronic Experiments: The unanesthetized guinea pigs which were exposed to 30-45 minutes of hypoxia—3% oxygen—had periods of apparent unconsciousness and heart irregularities. After removal from the chamber, the animal remained inactive for 15-20 minutes, and the pinna reflex was absent until activity was reinitiated. Subsequent exposures reproduced the same picture but sometimes micturition occurred. Prolonging the duration of exposure to 2 hours had a more pronounced effect upon the recovery. Activity did not appear for about 40-50 minutes; nystagmus was occasionally observed, and pinna reflex

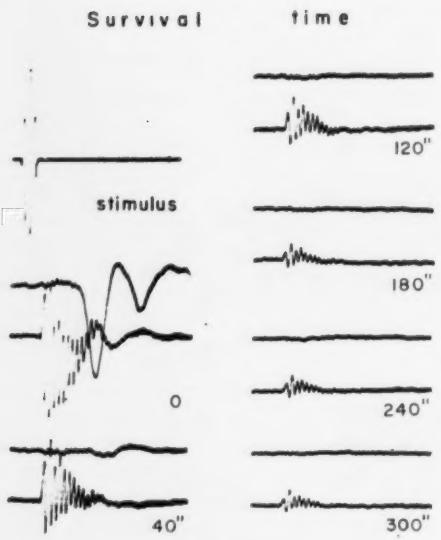


Figure 11

Fig. 11.—Guinea Pig A-57. This animal was exposed to 5 minutes of asphyxia. Time is measured in seconds from beginning of exposure. Survival time for action potentials was approximately 50 seconds. Microphonics were reduced to postmortem levels in approximately 150 seconds. Notice that these microphonics do not change in amplitude as asphyxia progresses. Stimulus, 8,000 cps tone-pips at 50 db. above threshold for action potentials.

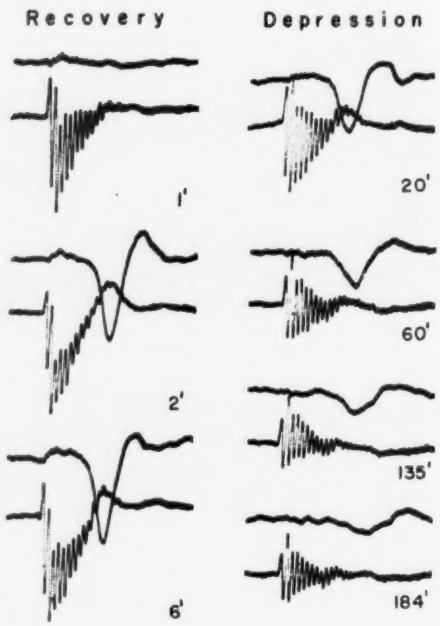


Figure 12

Fig. 12.—Recovery of cochlear responses of Guinea Pig A-57, after 5 minutes of asphyxia. Time is measured in minutes from the end of asphyxic period. The responses had fully recovered their normal amplitude in about 12-15 minutes. About 20 minutes after the asphyxia, disturbances in the heart beat appeared and continued for 3 hours of continuous observation. As soon as the heart beat irregularities occurred, the responses showed a progressive deterioration until almost complete extinction of action potentials and a significant reduction of microphonics amplitude had taken place. These changes are illustrated under the column "Depression." The animal was killed by intravital fixation 10 minutes after the last picture of the figure was taken. Photomicrographs of the organ of Corti, macula utricularis, and one crista ampullaris of this animal are presented in Figure 13.

was either absent or diminished for several hours.

These animals, in which hearing was tested before perfusion, showed cochlear microphonics and neural components comparable with those found in normal guinea pigs. In the histologic study of serial sections, no pathologic changes were found either in cochlear and vestibular receptors or in the central nervous system.

Comment

The depressant action of diallylbarbituric acid in urethan upon the respiratory center was clearly demonstrated in the experiments in which a gas mixture of nitrogen with 5% oxygen was used. The unanesthetized animals survived in it (at least 5 hours), while the anesthetized died within 2 hours. In the latter circumstance, the chemoreceptors were probably the main regulators of respiration. The increase in survival of anesthetized guinea pigs when respiration was controlled by means of a respirator further evidenced the depressant action of diallylbarbituric acid in urethan. This observation invalidates the suggestion, made in a previous report,[10] that this drug apparently did not depress the respiration of guinea pigs when their body temperature was lowered to 20 C.

The constant latency of CM during any type of oxygen deprivation supported the

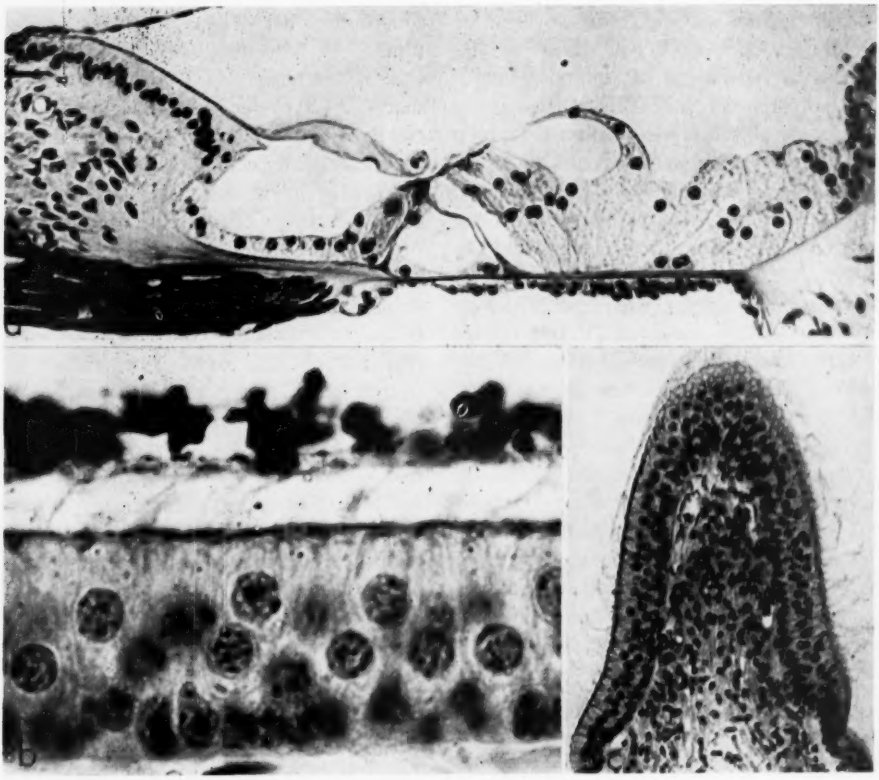


Fig. 13.—Guinea Pig No. A-57. (*a*) Normal organ of Corti (300 ×) at approximately 5 mm. from round window. The serial sections showed normal structures throughout both cochleas. (*b*) A section of macula utricularis (720 ×) showing normal structures. (*c*) Crista of the posterior semicircular canal (240 ×) showing normal structures. The other cristae as well as macula saccularis were found to be normal on both sides.

statement made in reports of hypothermia experiments [10,11] that the mechanism which precedes the generation of CM is insensitive to impaired metabolism. The increase in latency of neural components probably represents a successive block of nerve fibers.

The rapid change in the steady state of cochlear microphonics and neural components during oxygen deprivation, showed—once more—their dependence on the oxidative metabolism. Gas mixtures of nitrogen with 5% oxygen were (in the guinea pig) noxious to the cochlear responses. The maximum concentration of oxygen in the gas mixture which is necessary to produce a change in the steady state of CM, N_1, or N_2 was not determined in these experiments. In the cat, as demonstrated by Wever et al.,[2] the maximum for CM was 4% oxygen.

The survival of cochlear responses may be considered a relative measurement of the oxygen requirements of their generators. Under our experimental conditions this index depends primarily as follows: (1) in asphyxia, on the oxygen available in blood and lungs at the point in the respiratory cycle at which the trachea was clamped; (2) in fulminating anoxia, on the oxygen available in blood only, because the respirator probably replaced most of the lungs' air by pure nitrogen in few strokes; (3) in hypoxia, on the concentration of oxygen in the gas mixture.

Other factors on which the survival of cochlear responses depended are the oxidizing reserve, the metabolic rate of generators, and the energy provided by reactions such as glycolysis. These factors have been studied extensively in the central nervous system [12,13] by occlusion of the carotid and vertebral arteries. In this experiment the time from beginning of occlusion and the moment the responses of the region under examination disappear was called the survival time. It was considered to be an index of the factors mentioned before. In the cochlea, technical difficulties do not allow such determination yet, so that a comparison with the results obtained in the central nervous system cannot accurately be made. Nevertheless, the survival of N_1 and N_2 (90 and 60 seconds respectively) during asphyxia and fulminating anoxia, suggested that the oxygen requirements of the primary cochlear neurons may be of the same order as some regions of the central nervous system.

The decay of neural components was accompanied by a simultaneous depression of cochlear microphonics. The question was raised whether the decay of the former was due to those phenomena which underlie the anoxemia or to depression of CM which is accepted to be the stimulus of nerve fibers.[8] Probably both factors were involved. But, judging from the curves of Figure 2, anoxemia seemed to be the most important, because at the moment neural components were totally depressed, cochlear microphonics were reduced by half only. This observation indicated that the oxygen requirements of cochlear primary neurons was higher than that of the generators of cochlear microphonics.

The sharp break of CM curve (Fig. 2), about 300 seconds after asphyxia or fulminating anoxia were initiated, marked the boundary between CM_1 and CM_2. The former is the predominant component representing about 85% of CM. The cause of depression of CM voltage during impaired metabolism, acoustic trauma, and other conditions, has been discussed several times.[1,8,14] The theory of "variable resistance of cochlear microphonics" [8] proposed that CM_1 represents a modulation of the endolymphatic potential and/or intracellular potential of hair cells. According to this theory, the depression of CM_1 observed in our experiments may represent a depression of the source (EP and/or DC), or some alteration in the modulator mechanism, or depolarization of the boundaries of endolymphatic potential and/or intracellular potential of hair cells. Furthermore, the source of CM_1 depends on the state of its generators, which in turn depend on the oxidative reactions. All the factors mentioned may contribute to the decay of CM_1, but essentially, the phenomenon must be related to those chemical reactions which underlie the generation of energy.

The survival of CM_2 continued for hours after death.[15] It seemed to represent a direct transformation of acoustic energy into electric energy. The structures which are involved in this transformation are unknown. The decay of the potential was probably due to autolytic changes which are known to develop steadily after death.

The similar time-course decay of cochlear responses in both asphyxia and fulminating anoxia suggested that hypercapnea, vascular changes, and accumulation of metabolites associated with asphyxia, did not play an important role.

The recovery of cochlear responses may be an indication of the speed of physicochemical reconstruction of (1) the generators of endolymphatic potential, (2) intracellular potential, (3) of their boundaries, (4) of the modulator of CM_1, and (5) of the cell membrane of nerve fibers. It is known that the endolymphatic potential recovers in a few seconds.[16] This fact explained the recovery of CM_1 within 10 seconds. Reconstruction in nerve fibers seemed to be a fast event also. The delay observed in N_2 recovery is most likely related to time required for full reconstruction of some properties of nerve fibers, such

as refractory period and local excitatory process. There are indications that many factors, such as temperature, and duration and repetition of exposures of oxygen lack, modified the recovery period.

The overshooting of neural components seemed to be of the same type as that observed in other nerves after anoxia.[17] However, the fact that overshooting of summating potential followed the same time course as N_1 raised the question whether these two potentials are related, as had been proposed by Davis.[8] This matter is now under investigation.

The method of Bornschein and Gernandt[5] for selective removal of neural components from cochlear microphonics seemed to be efficient in the sense that N_1 and N_2 may completely be abolished. However, CM was also largely depressed.

The finding that the cytoarchitecture of the inner ear was normal in animals exposed to acute and prolonged oxygen deprivation, was in agreement with the observations of Falbe-Hansen et al.[4] In the acute experiments, where depression of cochlear responses was obtained for several hours, most likely some changes in structures and fluids did occur. At this stage, histochemical methods and electron microscopy may make possible the detection of such changes, even though hematoxylin-eosin stain and observation under light microscopy showed no abnormalities.

The absence of injury in the inner ear, and the apparently normal cochlear responses found in the chronic animals, suggested that the depression observed in the acute experiments was a reversible phenomenon.

Summary

In this investigation the survival of guinea pigs, behavior of cochlear responses, and histological studies of the inner ear were made following exposure to various conditions of oxygen deprivation.

Anesthesia with diallylbarbituric acid (Dial) in urethan (ethyl carbamate) considerably diminished the survival of guinea pigs exposed to an atmosphere of low oxygen concentration. This observation indicates that this anesthetic is a depressor of the respiratory center.

The dependence of cochlear responses on an adequate oxygen supply was demonstrated. Neural components (N_1 and N_2) were the most sensitive; their survivals were 90 and 60 seconds respectively when asphyxia or fulminating anoxia was used, while cochlear microphonics (CM_1) survived about 300 seconds. Depression of CM_1, N_1, and N_2 seemed to be related to those chemical reactions which underlie the generation of energy.

The recovery of cochlear responses after a bout of oxygen deprivation followed a definite order; CM_1 recovered within 10 seconds. N_1 lagged CM_1 by a few seconds, and N_2 was the last potential to reappear. Recovery seemed to depend upon factors such as duration and degree of oxygen lack, and probably is related to the reconstruction properties of the generators of cochlear potentials.

Exposures to asphyxia repeated at 1-hour intervals produced an apparent permanent depression of both cochlear microphonics and neural components, provided the duration of each bout is at least 3 minutes. In these animals no changes in the inner ear structures were found after hematoxylin-eosin stain and study with light microscopy. Unanesthetized guinea pigs exposed several times (30-45 minutes each) to gas mixtures of nitrogen with 3% oxygen and kept alive for about six weeks, also showed no histologic changes in the inner ear. Similarly, the temporal bones of animals exposed once for a period of 2 hours to the same gas mixture, and kept alive for several days gave no evidence of histologic change.

Routine histologic methods such as hematoxylin-eosin stain and light microscopy seemed to be inadequate for detecting changes in the inner ear structures of animals with apparent permanent depression of cochlear responses induced by repeated exposures to asphyxia. Chronic experi-

ments suggested that this depression was a reversible phenomenon.

950 E. 59th St.

REFERENCES

1. von Békésy, G.: Gross Localization of the Place of Origin of the Cochlear Microphonics, J. Acoust. Soc. America 24:399-409, 1952.

2. Wever, E. G.; Lawrence, M.; Hemphill, R. W., and Straut, C. B.: Effects of Oxygen Deprivation upon Cochlear Potentials, Am. J. Physiol. 159:199-208, 1949.

3. Lawrence, M., and Wever, E. G.: Effects of Oxygen Deprivation upon Structures of the Organ of Corti, A. M. A. Arch. Otolaryng. 55:31-37, 1952.

4. Falbe-Hansen, J.; Christensen, E.; Gisselsson, L.; Hansen, H. J., and Permin, P. M.: Effect of Acute and Prolonged Oxygen Deprivation on the Organ of Corti in Guinea Pigs and Cats, A. M. A. Arch. Otolaryng. 67:71-77, 1958.

5. Bornschein, H., and Gernandt, B.: Selective Removal of the Nerve Discharge Component from the Cochlear Potential During Anoxia, Acta physiol. scandinav. 21:82-89, 1950.

6. Fernández, C.: Effect of Oxygen Lack on Cochlear Potentials, Ann. Otol. Rhin. & Laryng. 64:1193-1203, 1955.

7. Tasaki, I.; Davis, H., and Legouix, J. P.: Space-Time Pattern of Cochlear Microphonics (Guinea Pig), as Recorded by Differential Electrodes, J. Acoust. Soc. America 24:502-519, 1952.

8. Davis, H.: Biophysics and Physiology of the Inner Ear, Physiol. Rev. 37:1-49, 1957.

9. von Békésy, G.: DC Resting Potentials Inside the Cochlear Partition, J. Acoust. Soc. America 24:72-76, 1952.

10. Fernández, C.; Singh, H., and Perlman, H.: Effect of Short-Term Hypothermia on Cochlear Responses, Acta oto-laryng., to be published.

11. Kahana, L.; Rosenblith, W. A., and Galambos, R.: Effect of Temperature Change on Round-Window Responses in Hamsters, Am. J. Physiol. 163:213-223, 1950.

12. Gerard, R. W.: Anoxia and Neural Metabolism, Arch. Neurol. & Psychiat. 40:985-996, 1938.

13. Sugar, O., and Gerard, R. W.: Anoxia and Brain Potentials, J. Neurophysiol. 1:558-572, 1938.

14. Eldredge, D. H.; Covell, W. P., and Davis, H.: Recovery from Acoustic Trauma in the Guinea Pig, Laryngoscope 67:66-84, 1957.

15. Wever, E. G.; Bray, C. W., and Lawrence, M.: Nature of Cochlear Activity after Death, Ann. Otol. Rhin. & Laryng. 50:317-329, 1941.

16. Davis, H.: Symposium on blood circulation: Summary of the Symposium and Comments, Tr. Am. Otol. Soc. 42:220-236, 1955.

17. Lorente de Nó, R.: A Study of Nerve Physiology, New York, The Rockefeller Institute for Medical Research, 1947.

Congenital Stenosis of the Nasal Passages

Report of One Case in an Infant Who Also Had Crouzon's Disease

COL. BYRON G. McKIBBEN (MC), U. S. Army

Bilateral congenital stenosis of the nasal passages may be severe enough to cause death from asphyxia soon after birth. If the infant overcomes this difficulty it may become emaciated and finally die of starvation because of inability to nurse and breathe through the mouth at the same time. The paucity of reference to this condition in medical literature leads one to conclude that this development anomaly is exceedingly rare.

Report of Case

A female infant, born at full term, Jan. 19, 1956, was observed to be in severe respiratory distress, because of inability to breathe through the nose. Her color was extremely dusky and she was gasping, with deep subcostal retractions. Intermittent crying, with improved respiratory exchange through the open mouth, was accompanied by marked improvement in color. With this improvement in airway the infant attempted to breathe quietly. The tongue, however, would press up against the palate and obstruct the breathing. This cycle of dyspnea followed by quietude continued because an oral airway was not provided.

Bilateral choanal atresia was suspected by the pediatrician.* An attempt to pass a No. 8 French catheter was met with obstruction just inside the nasal vestibules. It was noted that there was a very slight amount of air passing through the nasal passages, just enough to cause the accumulated mucus to form into bubbles at the nares, but not enough for respiration.

It was further noted that this pregnancy was complicated with polyhydramnios and there was threatened abortion at nine weeks' gestation. The head measurement of 13 ⅞ in. (35.3 cm.) was the upper limit of normal for a 6 lb. 10 oz. infant whose chest measurement was 13½ in. (34.3 cm.). The anterior fontanelle was large (3×3.5 cm.) and full, but not tense. The posterior fontanelle was large (1×1 cm.) and just anterior to this there was a large defect (2×2 cm.) representing a third fontanelle. There was a peculiar abnormality to the facial appearance; the eyes were wide-set and prominent, with mild divergent squint; the nose was unusually small with markedly depressed bridge; there was some prognathism and a tendency for the tongue to protrude. The impression was that of a craniofacial anomaly with narrow nasal passages, possibly incomplete atresia of the choanae.

When the infant was 2 days of age, examination revealed cardiac dilatation and marked cyanosis. Cyclic dyspnea was no longer observed. Apparently the child was too exhausted to continue struggling for breath.

It was at this stage that I first saw this infant. A silver probe was passed along the floor of each nasal passage. At the choanae firm resistance was encountered. The probe was forced through, giving the sensation of passing through a small opening in bone. When the probe was withdrawn, there was no blood stain, giving the impression that membrane had not been perforated. A No. 5 French ureteral catheter was then passed, against resistance, through the choanae and was observed in the pharynx. An attempt to see the choanae with a nasopharyngoscope, as suggested by Phelps[1] and Mayer,[2] was unsuccessful. An infant oral airway was inserted, with immediate improvement in breathing, and the color became a healthy pink. The metal airway was later replaced with a soft rubber tube which was held in place with adhesive.

Reexamination of the heart revealed that it was of normal size.

While the nasal passages appeared narrow anteriorly, it was felt that if air could pass more freely through the choanae, the anterior part of the nasal passages would open up. A tentative diagnosis of incomplete bilateral choanal atresia was therefore made.

Feeding was accomplished by stomach tube, and when the baby was 4 days old tracheotomy was performed.

Submitted for publication April 18, 1958.

From the Otolaryngology Service, Department of Surgery, Tokyo U. S. Army Hospital, San Francisco. Col. McKibben is currently stationed at William Beaumont Army Hospital, El Paso, Texas.

*Capt. Dennis N. Marks. Dr. Marks is now located at 2740 Fulton Ave., Sacramento, Calif.

During the following month, in spite of frequent suctioning, there were intermittent attacks of respiratory distress caused by mucus in the trachea. Nutrition, however, was satisfactory and the baby became stronger.

On Feb. 23, 1956, under general anesthesia, a palatal flap, including the palatine vessels, was cut according to the technique described by Owens,[3] and approximately 1 cm. of the posterior portion of the bony palate was removed with biting forceps. The nasal passages were found to be extremely narrow. The mucosa covering the vomer came in contact with that of the lateral walls. The septum in the region of the vomer was found to be approximately 5 mm. wide. This consisted of the cartilaginous septum which measured approximately 3 mm. in diameter, the two very thin plates of bone which eventually fuse to form the vomer,[4] and the mucosal coverings. This portion of the septum, including the mucosa, was removed as is usually done in the correction of choanal atresia. Following this a No. 8 French catheter was passed through each nasal passage into the pharynx. The catheters were cut external to the nares and sutured together. The palatal flap was sutured with black silk.

The immediate postoperative period was uneventful but later became quite stormy with the development of purulent tracheal secretion after one week, a convulsion followed by a period of apnea on March 6, and bronchopneumonia on March 13.

Nasal breathing, however, became satisfactory, and the tracheal tube was removed on March 28.

Rhinitis, with nasal congestion and increased flow of mucus was a later development. In order to allow the baby to breathe through the nose while nursing, nasal suction, followed by shrinkage with 0.25% phenylephrine hydrochloride (Neo-Synephrine), was done just before each feeding with a fair degree of success.

On April 12 there was a period of labored respiration and limpness, and on April 28 a period of apnea followed by noisy breathing and flaccidity of the limbs.

Hydrocephalus had been suspected for some time. Failure, however, to tap the ventricles with a needle inserted to a depth of 2½ in. (6.3 cm.) ruled out this possibility.

By May 10 the baby had improved enough to go home. A special nurse was employed, and a suction apparatus was rigged up on the vacuum cleaner.

Improvement continued, but when the baby was 6 months old premature closure of the cranial sutures was suspected and roentgenography confirmed premature closing of the coronal suture. A diagnosis of Crouzon's disease was made. The family returned to the United States when the baby was 7 months old, and further follow-up was unsatisfactory. It was learned, however, through a friend of the family, that the child continued to improve, and when 13 months of age she was able to walk by holding onto objects. She underwent three operations, the nature of which was not stated, and sometime during the summer of 1957 (age 18-20 months), she died. I was unable to find out the cause of death.

Comment

The symptoms in this case were similar to the cyclic dyspnea of bilateral congenital choanal atresia, described by Ronaldson[5] in 1881 and by Richardson[6] in 1914. In a previous publication[7] I pointed out that it might be difficult to differentiate between severe stenosis of the nasal passages and bilateral choanal atresia. In either case the infant may be able to force a little air through the nostrils, just enough to cause the nasal mucus to form into bubbles at the nares, but not enough for respiration.

At the time of operation, it was thought that the nasal septum, in the region of the vomer, was much wider than normal. Schaeffer, however, pointed out that the normal thickness of the cartilage lying between the two plates of developing vomer is 3 to 3.5 mm. He referred to Figure 43 in his text. This illustration, just under half size (× 0.45), depicts the osseous elements of the head in a fetus at term. The vomer clearly shows its formation in two plates. The space between these two plates, occupied by the cartilaginous nasal septum, measures 1.5 to 1.75 mm. in the illustration. Multiplying this reduced space by 2, the actual thickness of the cartilage is 3 to 3.5 mm.[4,8] Nasal obstruction in this case, then, was the result of a very small nose with the lateral walls lying too close together.

Craniofacial dysostosis of Crouzon represents a localized premature synostosis, causing retardation in the growth of the maxilla, some prognathism, ocular hypertelorism, exophthalmos with divergent strabismus, a peculiarly shaped nose, often described as resembling a parrot's beak, and increased intracranial pressure, with eventual loss of sight and mental deficiency. This type of dysostosis represents a variation of crani-

ostenosis, and, according to Greig's classification, it is a true oxycephaly.[9-11] It is believed that the case herein presented had the signs of this condition, and it can be assumed that the three operations the patient underwent were linear craniotomies for the purpose of opening up the prematurely closed skull sutures. It is surprising that stenosis of the nasal passages has not been described in other cases of Crouzon's disease.

Summary

Congenital stenosis of the nasal passages, when bilateral, in the newborn, is a rare anomaly that may produce the same type of cyclic dyspnea that has been described in cases of bilateral congenital choanal astresia.

One case, in an infant, with craniofacial dysostosis of Crouzon is presented and briefly discussed.

REFERENCES

1. Phelps, K. A.: Congenital Occlusion of the Choanae, Ann. Otol. Rhin. & Laryng. 35:143-151 (March) 1926.

2. Mayer, E., in discussion of Richardson, C. W.: Congenital Occlusion of the Postnasal Orifices, with Report of a Case, Tr. Am. Laryng. A., 35th Annual Meeting (May 5-7), 1913, pp. 226-231.

3. Owens, H.: Observations in Treating 7 Cases of Choanal Atresia by Transpalatine Approach, Laryngoscope 61:304-319 (April) 1951.

4. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920, pp. 41, 42.

5. Ronaldson, T. R.: Note on a Case of Congenital Closure of the Posterior Nares, Edinburgh M. J. 26:1035-1036, 1880-1881.

6. Richardson, C. W.: Congenital Atresia of the Postnasal Orifice, Lancet 2:439-444 (Aug. 15) 1914.

7. McKibben, B. G.: Congenital Atresia of the Nasal Choanae, Laryngoscope 67:731-755 (Aug.), and Errata, 986 (Oct.) 1957.

8. Schaeffer, J. P.: Personal communication to the author.

9. Reeves, D. L.: Craniofacial Dysostosis of Crouzon—The Surgical Treatment of Oxycephaly, in Brenneman's Practice of Pediatrics, edited by I. McQuarrie, Hagerstown, Md., W. F. Prior Co., 1957, Vol. IV, Chap. 25A, p. 1.

10. Burch, E. P.: Hereditary Craniofacial Dysostosis (Crouzon's Disease), in Brennemann's Practice of Pediatrics, edited by I. McQuarrie, Hagerstown, Md., W. F. Prior Co., 1957, Vol. IV, Chap. 38, pp. 64, 65.

11. Holt, L. E., Jr., and McIntosh, R.: Pediatrics, Ed. 12, New York, Appleton-Century-Crofts, Inc., 1953, pp. 784, 785.

Evaluation of the Sierra-Sheldon Tracheotome

LIEUT. COL. H. HASKELL ZIPERMAN (MC), U. S. Army, and CAPT. SPENCER P. THORNTON (MC), Army U. S.

Emergency tracheotomy is life-saving when performed for respiratory obstruction not correctible by nonsurgical means. As such, it is a procedure which should be within the capabilities of every physician. We questioned more than 1,500 medical officers newly inducted into the U. S. Army and found that approximately 50% of them either had not seen or had not done a tracheotomy. These physicians represent a cross section both of the graduates of all of the medical schools in the United States and practitioners of most of the medical specialties. In order to avoid an unnecessarily high mortality rate from acute respiratory obstruction incident to either war or disaster, it is essential that all physicians be taught to do tracheotomies. With the initiation of a practical method for teaching the principles of combat surgery to physicians[1] at the Army Medical Service School, Brooke Army Medical Center, Fort Sam Houston, Texas, all medical officer students are taught to do tracheotomies. The teaching is based on procedures practiced on laboratory animals. However, many of these physicians are not surgically oriented and are able to perform this procedure only with close and detailed supervision. It has become obvious that a simple, relatively foolproof means for accomplishing a rapid emergency tracheotomy, utilizing a minimal amount of equipment and supervision, needs to be found.

It is known that with thermonuclear warfare there will be a tremendous disparity between total casualties and medical capabilities. To overcome this disparity, consideration is presently being given to training auxiliary medical personnel such as dentists, nurses, veterinarians, and others in the performance of certain life-saving procedures. These trained people, acting under the supervision of a physician, will be expected to exercise little or no medical or surgical judgment in practicing a life-saving procedure. Instead, their function will be the application of a learned mechanical skill toward producing a desired technical result. In effect, their use should serve to provide a surgeon with six or eight "pairs of hands" to care for that many patients simultaneously and should prove to be both time-saving and life-saving. Any method which permits the mechanical production of an airway, provided it requires little if any knowledge of the detailed anatomy of the region, might lend itself to being utilized by auxiliary medical personnel in the performance of this life-saving measure. It was decided to investigate the Sierra-Shelden Tracheotome[2] as a device which might permit inexperienced operators to perform successful emergency tracheotomies.

Submitted for publication April 22, 1958.

From the Wound Studies Laboratory, Department of Military Medicine and Surgery, Army Medical Service School, Brooke Army Medical Center, Fort Sam Houston, Texas.

Materials and Methods

The standard laboratory animal used in the Wound Studies Laboratory of the Army Medical Service School is the female mohair goat, whose weight is between 80 and 100 lb. Anesthesia is produced with allobarbital-urethan (Ciba brand veterinary Dial-Urethane) given intravenously into an external jugular vein. This laboratory procedure is preceded by a group demonstration in which an instructor shows all details of the Sierra-Shelden Tracheotome and its use on an anesthetized animal. Immediately thereafter students proceed with their tracheotomies. When necessary, help is provided by qualified physician instructors. Success or failure as well as time per procedure and complications encountered are recorded. In addition to this, information is obtained from medical officers in regard to their specialties and whether or not they have previously done a tracheotomy. The occupational specialties of the nonphysician students are also recorded. Because the average length of a goat's trachea from thyroid cartilage to the superior border of the sternum is 23.4 cm., as many as four tracheotomies per animal are sometimes performed.

The Sierra-Shelden Tracheotome was developed to shorten the time required to perform an emergency tracheotomy and thus to lessen the trauma of a surgically performed tracheotomy to a critically ill patient. This tracheotome consists of a short, slotted 13-gauge needle, a tapered trache-

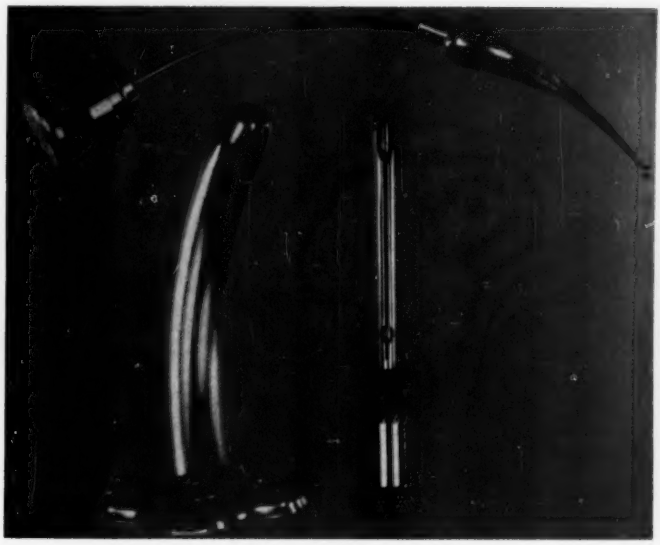


Fig. 1.—Sierra-Shelden Tracheotome, consisting of a short, slotted 13-gauge needle, a tapered tracheotomy tube, and a cutting trocar.

otomy tube, and a cutting trocar (Fig. 1). When using the tracheotome, the operator stands at the head of the patient, who lies with neck extended. The trachea is fixed between thumb and forefinger of the operator's left hand as the needle is inserted at a 45 degree angle percutaneously into the trachea at least one ring below the cricoid cartilage. The needle can be felt entering the tracheal lumen. Its proper position is verified by air passing through it with respiration. The needle is then rotated 90 degrees, so that the slot is turned lateralward (Fig. 2). The ball tip on the cutting trocar (already locked within the tracheotomy tube) is then engaged in the slot of the needle, and the entire instrument is pressed firmly into the trachea (Fig. 3). The needle must be held firmly, so that it acts as a track for the passage of the instrument, and is removed as the ball tip of the trocar passes beyond the sharp end of the needle within the trachea. The tracheotome is then pushed into place, and removal of the trocar with insertion of a plastic sleeve completes the tracheotomy (Fig. 4). No other instruments are required.

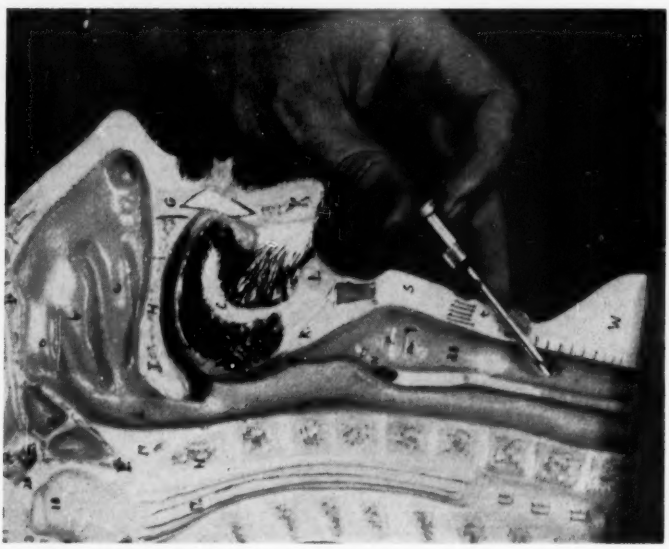


Fig. 2.—Slotted needle within the tracheal lumen.

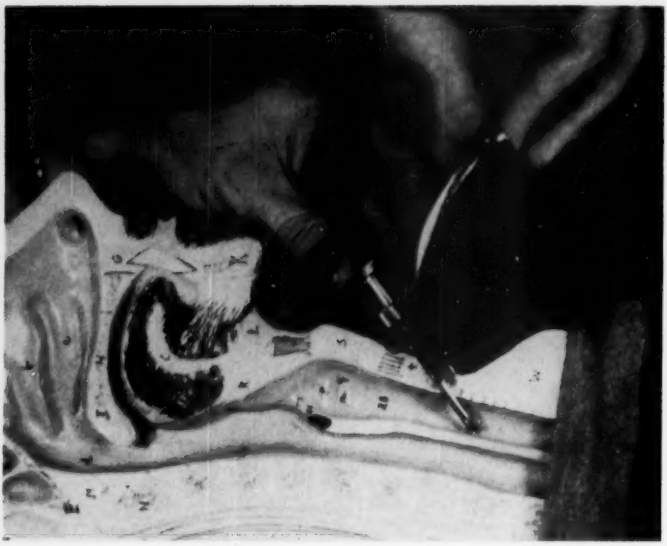


Fig. 3.—Tracheotome engaged in the slot of the needle.

Results

A total of 1,135 tracheotomies were performed on goats with the Sierra-Shelden Tracheotome (Table). Of these, 864 were performed by medical officers. Of the 864 tracheotomies, 149 were done by physicians with surgical training, whose average elapsed time per procedure was 31.9 seconds. The average operating time for 715 physicians without surgical training was 37.6 seconds. In 753 cases the trachea was removed and examined post mortem. Among these, there were 14 posterior tracheal perforations complicating the 561 tracheotomies performed by physicians. Several false passages with the instrument coming to rest in the subcutaneous tissues were immediately recognized and corrected by successful insertion of the tracheotome. Not a single instance of damage to the great vessels or other vital neck structures was recorded. All tracheotomies were successfully completed.

Among the 1,135 tracheotomies, 271 were performed by nonmedical personnel, including 155 by

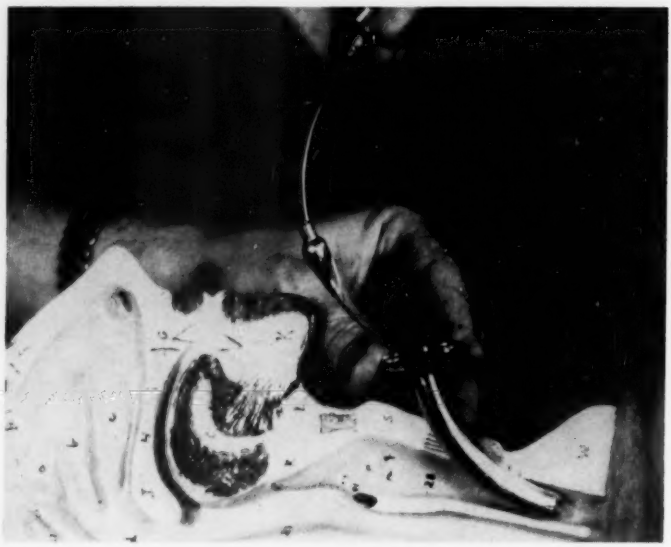


Fig. 4.—Tracheotomy tube within the tracheal lumen and the trocar being withdrawn.

Tracheotomies Performed

By Medical Officers			By Auxiliary Medical Personnel		
Specialty	No. of Tracheotomies	Average Elapsed Time, Sec.	Occupation	No. of Tracheotomies	Average Elapsed Time, Sec.
Surgeons	149	31.9	Nurses	155	42.6
General practitioners	487	37.0	Dentists	61	39.1
Internists	81	37.2	Enlisted men & medical service officers	55	43.5
Miscellaneous specialties	147	38.5			
Total	864	36.4	Total	271	41.9
In 561 tracheas examined, 14, or 2.5%, showed posterior tracheal perforations			In 192 tracheas examined, 8, or 4.2% showed posterior tracheal perforations.		

nurses, 61 by dentists, and 55 by medical enlisted men and Medical Service Corps officers. The average time consumed per tracheotomy was 42.6 seconds for the nurses, 39.1 seconds for the dentists, and 43.5 seconds for the enlisted men and Medical Service Corps officers. These auxiliary medical personnel produced posterior tracheal perforation in 8 of 192 tracheas examined. Of these six were produced by the nurses and two by the combined enlisted men and Medical Service Corps officers. Although all tracheotomies were successfully completed, some required a second attempt because of a false passage produced on the first try. No perforations of large neck vessels or other vital structures were recorded.

Comment

An emergency tracheotomy performed in an operating room where surgically experienced personnel are available presents no problem in accomplishment. But what of the patient with acute respiratory obstruction who requires a life-saving tracheotomy in the hospital emergency room or on the ward? How about the victims of disaster, either natural or thermonuclear, who will be treated in hastily organized treatment facilities by physicians quickly mobilized from the local area? Of our 864 medical officers 48% had never done a tracheotomy and could not be expected to do one by conventional means in time to save a patient's life. A special problem exists in the combat zone in that most casualties who require tracheotomy as a life-saving procedure are neither in a medical installation nor where a physician can be expected to perform the required life-saving surgery. Any device which is simple to use, requires little or no knowledge of anatomy, and needs no additional apparatus lends itself particularly well to overcoming the difficulties outlined above.

Performance of tracheotomies in goats is generally easier of accomplishment than in humans because of the more superficial location of the trachea, the greater mobility of the goat's neck, and the greater length of the goat's trachea. On the other hand, the smaller diameter of the average goat trachea (1.75 cm. $\times$ 2 cm. as compared with 2 cm. $\times$ 2.25 cm. for the average human adult) renders insertion of the Sierra-Shelden tracheotomy tube more difficult. Additionally, thickness and toughness of the goat's skin covered with its mat of hair makes insertion of both needle and trocar more hazardous from the standpoint of producing perforation of the posterior tracheal wall. Taking all of these factors into consideration, it is significant to note that all 1,135 tracheotomies were successfully accomplished and in an emergency would have resulted in saving the lives of casualties with respiratory obstruction requiring tracheotomy. Several false passages were produced, with the tracheotome coming to rest in the subcutaneous tissues of the neck. In none of these instances was any injury to the great vessels or to lateral neck structures produced. The false passages were immediately recognized and were followed by successful tracheotome insertion. In spite of these false passages, the average elapsed time per tracheotomy was less than 40 seconds for all physicians. Unlike the surgically performed tracheotomy, bleeding from pretracheal and tracheal vessels has presented no problem in this series. In the human patient in whom the thyroid isthmus traverses the field, the needle and trocar can be inserted either cephalad or caudad to this structure to avoid troublesome bleeding. The other small vessels perforated by this instrument are apparently successfully occluded because the tapered shape of the cannula causes it to fit snugly into the tissues traversed.

The question as to whether training in the use of the Sierra-Shelden tracheotome should be afforded to all medical and auxiliary medical personnel is a knotty problem. On the one hand, we know that an airway can thus be produced in all instances in 45 seconds or less no matter how inexperienced the operator. On the other hand, experience gained in this series shows that in 22

of 753 tracheotomies performed, or 2.92%, perforations of the posterior tracheal wall resulted. Of these, 2.5% resulted from the efforts of physicians and 4.2% from the efforts of auxiliary medical personnel. Unfortunately there are no figures available in regard to how many lives are lost as a result of poorly performed emergency tracheotomies for patients in acute respiratory distress. It is our feeling that this instrument simplifies and speeds up the production of an airway for the patient who requires tracheotomy as an emergency procedure. As such it would be most helpful as an aid to inexperienced personnel who have done few or no tracheotomies in the past. Unquestionably, if available in all hospital emergency rooms and on wards, its use would be responsible for saving lives. In preparing for a mass casualty situation, such as might result from either thermonuclear or natural disaster, consideration should be given to training auxiliary medical personnel in the use of this instrument. A significant decrease in the mortality from traumatic upper respiratory obstruction would probably result. The complications arising from posterior tracheal perforations could be cared for later as symptoms and signs are manifested and after the patient had recovered from the anoxia incident to the obstruction. One author* who has performed 10 tracheotomies on human patients with this instrument states that the technique is so simple he recommends its use for both emergency and elective tracheotomy.

Summary

An evaluation of the Sierra-Shelden Tracheotome as an instrument for performing emergency tracheotomy was conducted in the laboratory in order to determine its value for use by inexperienced personnel in emergency rooms, hospital wards and in the field for combat casualties or disaster victims. Of 1,135 tracheotomies performed, 864 were done by physicians and 271 by auxiliary medical personnel. All were successfully completed in an average time of 45 seconds or less. The only significant complication was posterior tracheal perforation in 2.92% of the cases.

Army Medical Service School, Fort Sam Houston, Texas.

REFERENCES

1. Ziperman, H. H.: A Method of Teaching Combat Surgery, J. M. Educ. 31:746-750 (Nov.) 1956.

2. Shelden, C. H.; Pudenz, R. H.; Freshwater, D. B., and Crue, B. L.: New Method for Tracheotomy, J. Neurosurg. 12:428-431 (July) 1955.

3. Crue, B. L.: Use of the Shelden Tracheotome in Military Medicine, Armed Forces M. J. 7:1193-1195 (Aug.) 1957.

Progress in Otolaryngology

Otitis Media and Complications

Summaries of the Bibliographical Material Available for 1957

B. R. DYSART, M.D., Pasadena, Calif.

The literature on otitis media and complications for 1957 contained numerous interesting articles on the operations for chronic otitis media and the reconstruction procedures for improvement of hearing damaged by infection.

Anatomy

Two apparently conflicting theories on the pneumatization of the temporal bone are given in the first two articles.

Tumarkin [1] (Liverpool) found that the children living in the slums of Liverpool have smaller mastoid development than the same age children coming from better homes. When he examined their drums he found pathology in 20% as compared to 5% in the middle class children. Audiograms were abnormal in about the same proportion. He concluded that hypocellularity is the result of respiratory disease ("rampant in the lower classes") which can inhibit normal cellular development by setting up a low-grade exudative tympanomastoiditis that is frequently symptomless.

He suggests that most cases of later developing cholesteatoma and possibly otosclerosis are due to this low-grade infection. He does not believe that the size of the mastoid is hereditary, as has been suggested by Cheatle, Diamant, and others.

(This is an article that will repay some study. It is 95 pages long, appearing in three succeeding months. The first month is concerned with the comparative anatomy of many of the vertebrates and contains many interesting ideas and illustrations.)

Submitted for publication Aug. 7, 1958.

Diamant [2] (Halmstad, Sweden) has examined over 1,600 persons otoscopically and by x-ray of the mastoids. Measurement of the size of the mastoid development was done by planimeter. The average size was found to be 12 sq. cm., with variations from 0 to 30 sq. cm. In the group were 275 patients with perforations of the drum.

He is convinced that heredity is the determining factor of the size of the air cell development. He has seen manifest infantile otitis which did not result in a small mastoid air cell development in the adult, and even seen large cell systems develop after mastoidectomies performed in early childhood.

He agrees with McKenzie and Rüedi that cholesteatoma precedes and causes marginal perforation. "The gradual development of a central perforation is a common observation in the course of otitis media. On the other hand, no investigator has reported observing the actual appearance of marginal perforations."

He has never found a marginal perforation in an ear showing an air cell system larger than 10 sq. cm.

He does not think that the small size of a mastoid is a causative factor in the perforations, but it seems to be of prognostic and diagnostic value.

(These theories are not contradictory but complementary. One article several years ago traced a similar pneumatic development of the mastoid through three generations. McMillan, of the x-ray department of the Massachusetts General Hospital, showed at the Los Angeles Research Study Club a series of x-rays of the mastoids of a nurse

who developed mastoiditis in a well-pneumatized mastoid. Operation was advised but refused. After several months the x-rays showed complete sclerosis in the formerly pneumatized mastoid.)

Diagnosis

Schuknecht and Trupiano [3] (Detroit) reported the very interesting diagnostic problem of a boy 16 years of age with dislocation of the incus resulting from bilateral mastoidectomy operations at the age of 2½ years.

Hearing had been badly impaired and he had worn a hearing aid for the year before the first examination. He stated that, after swimming two months before, he developed a left earache with purulent drainage for one week. He then noticed that when his head was moved to the erect position he would hear a click and the hearing would be greatly improved as long as the head was held upright. Audiograms showed that when the head was erect there was only a 5 db. loss at 2,048 and a 15 and 20 db. loss at 512 and 1,024. When the head was bent forward the hearing loss increased to 50 db. in the speech range. This hearing improvement began to disappear in a few weeks, and seven months later he was again wearing his hearing aid and the loss was stationary at an average of 50 db.

An exploratory operation was done, and the incus was found to be displaced downward. When rotated very slightly it made contact with both the head of the stapes and the handle of the malleus. The incus was easily forced upward into its normal position. Hearing after operation remained almost normal for four months and gradually lowered to a 30 db. level at nine months.

It was decided that the improvement of hearing after infection was due to formation of soft tissue that held the incus in proper position for a while. They plan to perform a myringostapediopexy on the opposite ear soon.

In the same article the problem of the patient with bilateral chronic mastoiditis was discussed. In cases where the patient's general condition is good they do operations on both ears at the same time if the combined operation does not take longer than four or five hours. This results in a saving of time and discomfort to the patient, and the end-results are just as good.

Pathology

Friedmann [4] (London) has published more evidence that the nonpneumatized mastoid is the result of chronic infection of the middle ear. He has examined 796 specimens of bone chips removed at mastoid operations between April, 1953, and August, 1956. The microscopic sections (reproductions are excellent) show alternate absorption and deposition of new bone during repeated attacks of infection.

Masked Mastoiditis

Parr and Hallberg [5] (Mayo Clinic) reported the case of an 8-year-old girl admitted because of unexplained fever of six weeks. She had had attacks of sinusitis and bronchitis since the age of 6 months. Six weeks before admission there was an acute otitis media for which she was given penicillin and later erythromycin. The ear had not drained and the condition had apparently subsided. Examination at admission showed only a dull thickened drum, but x-ray showed clouding of the mastoid. Myringotomy showed a completely dry ear, but mastoidectomy showed extensive destruction with granulations and pus over the wall of the lateral sinus. The sinus was compressible and was not opened. Recovery was rapid after the operation.

The case was diagnosed as "masked" mastoiditis. They think it is frequently found where initial antibiotic administration has been inadequate. It should be suspected in any case of unexplained fever, especially if there has been an earache at the onset.

Rosenwasser and Adelman [6] (New York) commented on the increased frequency with which serious complications of mastoiditis were being seen; frequently the symptoms have been so slight that pediatricians, general

physicians, and even otolarygologists have missed the diagnosis. They report three such cases, all with minimal symptoms but with serious lesions. A child of 5½ had a destructive mastoiditis, a large perisinus abscess and an extradural abscess with only a mild upper respiratory infection and no earache seven weeks before. The x-rays showed clouding of the right mastoid and no apparent destruction. The drum showed only slight thickening and there was some tenderness far back over the region of the lateral sinus. The second case was a man of 52 who had had slight pain in one ear eight weeks before operation. Four weeks later he developed dizziness, which was diagnosed as Ménière's disease by several physicians. Operation showed an extensively diseased mastoid with a fistula of the external semicircular canal and labyrinthitis. The third case was a 19-year-old boy with a chronic otitis media for seven years. He developed sudden symptoms of meningitis, but was treated for two weeks before an otologist was called in who recognized that the otitis was the cause of the meningitis. Operation found an infected cholesteatoma and extradural abscess. The patient died the next day, and autopsy revealed a large cerebellar abscess.

They found it "difficult to understand the almost dogmatic resistance of some experienced physicians to the fact that otitic complications are still occurring and require surgical intervention for their cure." They mention the frequency with which x-rays show slight cloudiness even when operation shows much destruction. (These three cases might also be classed as masked mastoiditis.)

Acute Otitis Media

Davison[7] (Danville, Pa.) in discussing antibiotic therapy stated: "With reference to the probability of preventing a mastoid operation, the patient with acute otitis media under the care of the average practitioner is worse off now than he was in 1946 in the early days of penicillin therapy. In an attempt to determine why, I have analyzed the charts of nine patients needing 'simple' mastoid operation in 1955 and 1956. The reasons may be summarized as follows:

1. Misplaced confidence in the effectiveness of glycerine ear drops.
2. Failure to employ early myringotomy.
3. Delay in use of antibiotic therapy until bone necrosis has occurred.
4. Inadequate amounts of antibiotic.
5. Organism not sensitive to the drug used."

He has been using tetracycline by mouth for early infections, but if there is no prompt response in 48 hours he uses fortified procaine penicillin by intramuscular injection with 1,000,000 units five times daily as the average dose for an adult.

A survey of cases of acute otitis media was conducted by the Working-Party for Research in General Practice[8] in England. The survey was done by 28 doctors in general practice for the year of 1955. There were 1,323 attacks recorded in 1,162 patients. (The total number of patients in their care was 47,661.) One of the surprising results of the survey was the fact that only 1% of the cases were referred to a specialist or to a hospital. In almost all cases there was a follow-up examination six months after first seen, and a surprisingly low number of complications were reported. There was one death from meningitis. Most of the cases were treated by oral antibiotics, and only 0.1% were treated by myringotomy.

Chronic Otitis Media and Mastoiditis

Sanderson,[9] (San Diego) in reviewing the conservative treatment of chronic otitis media, found that only 30% needed major surgery. In his series of 100 patients with an average drainage duration of eight years the remaining 70% were cleared up by attention to general health and local treatment using chiefly dry antibiotic powders along with other local treatments.

Goldstein[10] (New York) advises that when an infection of the ear does not respond to treatment by the antibiotic indicated by the sensitivity tests the treatment should be stopped for a few days and the ear recultured. In 14 such resistant cases (in 210

patients) the second culture showed a definite change, and different antibiotics were indicated by the sensitivity tests. He uses the pure powder of the antibiotic, using 20-40 mg. insufflated into the external canal twice weekly. All 14 resistant cases were cured.

Fritz [11] (Anchorage, Alaska) reported that chronic otitis media and mastoiditis is very common in the natives of Alaska, and that there is very inadequate provision for their care. He has done 149 mastoidectomy operations on 122 patients. He began by grafting most of the radical operations but soon found a large number that filled with pseudocholesteatoma. He found little difference in the healing time after giving up grafts. He has become convinced that postoperative treatment should be minimal, and no longer cleans cavities or cauterizes granulations. He uses only a powder of oxytetracycline (Terramycin), polymixin and, Timofax (a preparation containing 10% undecylenic acid). Most of his cavities are dry and healed in six weeks. He found the Alaska native notoriously susceptible to superficial skin infections. Impetigo is very common in children and adults. (A few years ago while on a trip to the northwest side of Vancouver Island I was told by a recent graduate of McGill Medical School that all the native Indian children that he saw had running noses and running ears. He attributed it to their poor diet of white flour and large amounts of sugar.)

Moore [12] (New York) reported his results in 50 cases of chronic otitis media treated by modified radical mastoidectomy during the past 10 years. He concluded that the presence of cholesteatoma was not a contraindication to the modified operation. Each case must be inspected carefully during the operation to decide how radical the operation should be. He preserved the matrix where it was found to be sealing off the middle ear and saw no complications afterward. His results in obtaining good hearing and dry ears were excellent. In five of his cases he allowed the head of the malleus to remain even when the incus was removed. Both Day and Walsh in their discussion thought that where the incus was no longer present the head of the malleus should be removed, both for the purpose of getting a better exposure of the attic and also to allow the drum to be laid in better contact with the head of the stapes. (His illustrations of the x-rays are good and the reproduced audiograms showing improvement in hearing after operation are impressive.)

Ohrt [13] (Odense, Denmark) reported on 34 revisions of radical mastoidectomy cavities that were done in the years 1939 to 1954. During this time 419 radical mastoidectomy operations had developed secondary cholesteatomas and the remaining 21 had osteitis and infected granulation tissue. One of the most frequent causes of trouble after the primary operation was the development of small narrow cavities that could not easily be cleaned. Information was not available in many cases as to whether skin grafts had been done primarily. One case that was known to have been skin-grafted had a large cholesteatoma under the graft. Only one case, a man of 25, ended fatally. There was a recurrent cholesteatoma; lateral sinus thrombosis developed, and the patient died of septic infarction of the lung. (The year was 1939, and, although Prontosil was used, the antibiotics were not available.)

Lee [14] (London) reported that of 7,416 men examined for military service during 1953-1954 3% were rejected for chronic otitis media. Although this is a smaller percentage than in preantibiotic days, it shows the need for careful treatment and follow-up of the acute cases to prevent chronicity.

It is interesting that the rate of rejections was twice as great in Glasgow as in the south of England. Since the standards were precise, it was thought that the variation was due to the variation in disease. Another study, the comparison of the number of hearing aids distributed to patients between the ages of 10 and 30, confirmed this opinion. Other studies showed that chronic otitis media "is still associated with schools not attended by the abler children, with

employment requiring little skill and with large families."

(These figures agree with Tumarkin's opinion that chronic otitis media and the resulting lack of mastoid pneumatization occurs much more frequently in the "artisan" class as contrasted with the "middle" class.)

Aschan and Nohrman [15] (Uppsala, Sweden) treated 33 cases of otosalpingitis with deep x-ray and reported good results in 30. All of these cases had failed to respond to the usual previous treatments. Their cases included 16 cases of chronic otitis media, and 3 were pilots who had repeated attacks of obstruction of their Eustachian tubes. They used from 600 to 800 r for each side in the bilateral cases. They are convinced that deep x-ray is much better than radium.

(Opinions vary as to the value of radiation in chronic otitis media and obstruction of the Eustachian tubes. It is being used much less in this country than formerly.)

Davison [16] (Danville, Pa.) reported results in 45 modified radical operations for chronic otitis media. He is sure that there would be fewer delays in advising early operations for chronic otitis if it were realized that a high percentage of these cases would have their hearing not only preserved, but frequently improved. He finds that the ears heal within an average of eight weeks without skin grafts. He does not find it possible to determine whether the long process of the incus is eroded if it is embedded in granulations, so he advises its removal if the hearing loss is as great as 25 db. and if erosion is suspected. In 12 cases in which the incus was destroyed by infection or removed at operation, the hearing remained at or above the 30 db. level.

Sixty-six per cent of his patients had a postoperative hearing level of 30 db. or better in the speech range.

Rohan [17] (London) reviewed the literature on artificial drum membranes and devised an instrument which he called the myringoplast to assist in placing the artificial drum over the perforation. He uses a thin sheet of polyethylene for his patches, especially for diagnosis, preferring it to the diagnostic probe, and found that many perforations would heal if kept patched for a few weeks or months. His myringoplast consists of a fixed outer tube with a flat end piece and an inner mobile wire, carrying two prongs which retract to release the patch when it is placed over the perforation.

Turner [18] (Odessa, Texas) and Dobson (Houston, Texas) discussed the use of immune globulin in treatment of 12 patients, all children who were almost continuously ill with respiratory infections. Many of these children "had been given antibiotics by the gross and vitamins by the carload." These children were given convalescent serum, 2 to 4 cc. each month. Their response was excellent in each case, all cases having been followed 16 months or more.

The γ-globulin measurements were done at Baylor University College of Medicine, using serum paper electrophoresis in which concentrations are reported as percentages of total serum protein present. The average for healthy adults is 15% with a normal range of 9%-20%. Their 12 cases ran γ-globulin of 15.6%, 17%, 13.2%, 12.3%, 14%, 10.1%, 11.4%, 10%, less than 1%, 10.2%, 13.5%, 5.1%.

γ-Globulin is formed in the reticuloendothelial system outside the liver, and this is where the antibiotics are found. The α-globulin, the α_2 and γ fractions are formed in the liver. (This treatment has proved to be very effective in the hands of many observers.)

Davison [19] (Danville, Pa.) reported results of his atticomastoidectomy operation on 45 ears during the period of three years. In that same period the complete radical operation was necessary for 77 cases. He tries, where the ossicular chain has been interrupted, to perform what Wullstein describes as his Type 3 tympanoplasty. He finds that cases with normal bone conduction and normally functioning windows

need not have a loss of more than 20 db. even after removal of the incus. In 12 cases with the incus destroyed by infection or removed at operation the hearing in the speech range remained at or above the 30 db. level. He does not skin-graft cavities routinely and advises preservation of the cholesteatoma matrix to hasten healing and help preserve hearing.

After removing the packing on the second or third day he applies plain boric acid powder to the cavity with a hand powder blower and has the patient continue this twice daily until the cavity is dry; this discourages granulations without impairing the regrowth of epithelium. In three cases the boric acid powder stopped discharge where Neo-Cortef drops (containing hydrocortisone and neomycin) had failed.

He disagrees with the following statements which have recently appeared in the literature: "If the ossicular chain is interrupted, hearing is likely to be better if the tympanic membrane is removed"; "40% of patients having a modified operation will eventually require a complete radical operation"; "useful hearing following the modified operation depends upon maintaining the integrity of the ossicular chain"; "it is best to leave a dislocated incus," and "a modified operation is never indicated when complications are present."

If after healing the drum is retracted and moves poorly, he inflates, does myringotomy, and aspirates mucus or serum; this is repeated if necessary. (A very concise article, showing much work and thought on the subject.)

Moore [20] (New York) reported his results in 50 consecutive cases of chronic otorrhea in which operation had been done by the endaural modified radical technique. In 42 of the 50 cases cholesteatoma was found. The average duration of drainage was 15 years. During the 10 years covered by this report there were seen also 47 cases in which disease was so extensive and the hearing was so far destroyed that a complete radical mastoidectomy was necessary. The choice between radical or modified radical operations was made at operation after inspection of the pathology with magnification and after good surgical exposure. He did not hesitate to leave part of the matrix of the cholesteatoma, especially the part sealing off the middle ear, but was especially careful in these cases to leave a wide cavity for postoperative observation. He found that the cavities with some matrix left behind healed more quickly and gave no more postoperative trouble than the cases that had been completely cleaned out and skin-grafted.

Careful audiometric comparison of pre- and postoperative hearing showed much improvement of hearing in some cases and conservation of hearing in the majority.

Tympanoplasty

Goodhill [21] (Los Angeles), in a symposium on deafness in children, described first the congenital anomalies and then discussed chronic otitis media with effusion, cases with persistent central perforations requiring myringoplasty, and cases of chronic purulent otitis media with necrosis of the ossicles. He is convinced that if serous otitis is neglected there is a probability of fibrous fixation of the ossicles and even necrosis of the lenticular process of the incus. "It is important, therefore, not to look benignly upon this problem and not to allow it to remain for any significant duration."

Chronic central perforations are repaired by split-thickness skin grafts. Chronic suppurative otitis media is no longer approached through preordained radical or modified radical mastoidectomy, but by exploratory mastoidectomy and exploratory tympanotomy through endaural and omega canal incisions. After these careful explorations a conservative removal of necrotic bone is done, combined, if necessary, with some kind of tympanoplastic procedure to conserve hearing. Cholesteatoma formation demands complete removal and the proper tympanoplastic reconstruction.

Zöllner [22] (Freiburg, Germany) discussed his special approach for tympanoplasty, which he calls meatoantrotomy. He uses this approach when there is a dry central perforation with a canal too narrow to operate through and also in infected cases where the antrum must be opened. The incision is similar to the old postaural incision but goes above the ear, allowing the whole upper ear to be laid down out of the way. The meatal wall is then incised to allow inspection of the middle-ear contents. After removal of any pathology, the edges of the perforation are freshened, and a medium thickness skin graft 0.5 to 0.6 mm. thick is used. (The illustrations should be studied as it is difficult to follow the details of the procedure. He reports excellent hearing improvement with this technique and gave a summary of his results in a later paper given at the Washington International Congress.)

Zöllner [23] (Freiburg, Germany), in the symposium on chronic suppuration of the temporal bone at the Washington International Congress of Otolaryngology, gave his ideas on prognosis of the newer tympanoplasty operations. He reported results of operations on 267 patients in four years. In cases with no break in the ossicular chain he got improvement in hearing in 90% of 52 cases. Twenty-eight per cent of all his cases had no hearing improvement. He at first operated in two stages in cases with active infection but now rarely finds this necessary, finding that the inflammation in cholesteatoma is relatively harmless, and that healing is rapid after removal of pathology.

Specimens removed at operation showed that cholesteatoma frequently develops in hernia-like invaginations of atropic scars of the tympanic membrane. In three cases cholesteatoma developed after skin grafts. In one illustration there are sebaceous glands, hair follicles, and a small cholesteatoma under a graft that was removed after one year. He is at present leaving a small acrylic tube 1 mm. in diameter in the Eustachian tube for three weeks in cases with obstruction of the tube.

Rambo [24] (New York) has devised a technique of plastic repair that is the same in all his cases. He does a complete radical operation in each case, turns a flap of the temporal muscle down over the middle ear; a fenestra is made in the horizontal semicircular canal, and skin flaps from the canal are laid down over the muscle. Three cases are reported, all obtaining serviceable hearing. The diagrams make the technique plain. He found that when he tried to conserve tissues of the middle ear there were frequently bits of cholesteatoma left behind that caused recurrences. His skin grafts were always followed by some fibrosis and adhesions that frequently destroyed a temporary gain in hearing.

(This certainly seems to be a technique worth trying where a complete radical procedure is necessary because of extensive pathology. If the average hearing obtained is as good as the three cases reported, this may well replace a number of the Class 3 and 4 tympanoplasties.)

Bell [25] (Roanoke, Va.) reviewed his tympanoplastic procedures and gave his results in 100 cases. In his earlier cases of myringoplasty he found that recurring perforations seemed to occur most frequently along the inferior margin of the drum. He assumed that the epithelial layer had not been thoroughly removed from this edge of the drum since this area is difficult to see well. By using more care in this area his results improved.

Pietrantoni and Bocca [26] (Milan, Italy) have repaired 33 cases of long-standing dry perforation of the drum by myringoplasty. They have had 94% good results with an average of 10 db. gain for speech frequencies. They found that in no case was the hearing loss greater than 30 db. when due to a perforation, and sometimes there was almost no loss. They found that posterior perforations were more apt to produce loss in the frequencies below 1,000

while anterior perforations were apt to cause loss in the frequencies above 2,000.

They perform myringoplasty only if the ear has been dry for one year, no visible pathology of the middle ear, and if there is a gain of at least 10 db. to 15 db. after a temporary closure of the perforation by a membrane or pledget of moistened cotton.

At operation a modified Lempert incision is used and a posterior flap turned down temporarily for better vision. The epithelial layer is then removed from the drum and the adjacent canal wall. A large full thickness or three-fourths thickness graft is then placed over the perforation and the denuded drum and canal. This large bed is the secret of good nourishment for the graft and their high percentage of "takes," almost 100%.

Juers [27] (Louisville, Ky.) again urged that we conserve hearing by using the modified radical technique with variations in technique as demanded by the pathology found. There should be no compromise with complete removal of pathology since serviceable hearing can now still be attained by one of the tympanoplasty techniques. "The basic requirements for effective air conduction after surgery include (1) an inflatable air space over the round window membrane, (2) contact of the tympanic membrane with the stapes if the ossicular chain is interrupted, and (3) an unimpeded stapes."

Pick [28] (Los Angeles) presented 10 typical cases treated by tympanoplasty with diagrammatic illustrations of the pathology and with audiograms to show the hearing impairment before and after operations. He has tried Zöllner's method of reopening a closed Eustachian tube by passing a small polyethylene tube through the Eustachian tube from the middle ear. Inside the plastic tube a silver thread was left for three weeks and both were then drawn out through the nose. The Eustachian tube remained open three months, but then the hearing gradually dropped without apparent reason.

Hall and Rytzner [29] (Sweden) have been trying a modified tympanoplasty in which they remove the stapes and use either the incus (preferably) or the malleus inserted directly into the oval window. A skin graft is then laid over in the hope of getting a columellar effect. They reasoned that birds require only one ossicle in the transmission of sound. At first they tried placing the columellar ossicle in contact with the foot of the stapes but they found that this rarely gave any prolonged improvement. They gave audiograms of three cases with much improvement of hearing, lasting in two cases for one year and in the third case for three months. They do not report the number of cases in which they have used this procedure or in what percentage of cases they have got good results.

Miodonski [30] (Krakow, Poland) has been doing a columellization operation since 1951, creating a small middle ear by means of free full-thickness skin grafts as well as pedunculated plastic flaps from the tympanic membrane. He hopes to get an adhesion between the plastic flap and the head of the stapes. Recently he has tried to free the long process of the malleus and connect it with the head of the stapes. He has had good results with this procedure in a small number of cases.

Beales [31] (Doncaster, England) reported his results in 17 cases of myringoplasty and 42 cases of tympanoplasty. Although simple repair of perforations by skin graft (myringoplasty) has been criticised by Wullstein on the grounds that one cannot adequately inspect the attic area, Beales has got excellent results in 70% of his cases. He does cases that have been dry for years and have no evidence of damage to the ossicular chain. He uses full-thickness grafts taken from behind the ear, exposes the drum by a modified Lempert incision, and is careful to strip the epithelium well up along the external meatus to be sure to provide a good bed for the graft. He urges that the edges be stripped from the canal toward the perforation; otherwise the perforation will be enlarged. If the perforation is large he places gelatin foam

to support the graft and prevent adhesions to the promontory.

In his tympanoplasty operations he uses Wullstein's classification and follows his techniques. Out of 42 tympanoplasty operations 20 cases showed marked improvement in hearing, and all except 5 had resulting dry ears; 14 cases had slight improvement in hearing; 5 had no change, and only 3 were worse than before operation.

Ormerod and McLay[32] (London) reported their results in 38 cases of operations on the sound-conducting apparatus. They found the greatest single cause of failure was a neglect of early inflation of the Eustachian tube. They now begin this on the 12th day and repeat it frequently. In all cases it must be shown before operation that the Eustachian tube is patent. Eradication of infection must be the most important objective and this must be attained both by antibiotics and by surgery, however radical it may have to be.

They divide their cases into four groups: (1) myringoplasty, in which a full-thickness graft is applied to a tympanic perforation; (2) epitympanomastoidectomy, where the ossicular chain is intact, and after removal of infection the defect is repaired by a full-thickness graft; (3) columella type tympanoplasty, in which the ossicular chain is interrupted and the graft is made to lie on the head of the stapes. The contact of the graft with the stapes is best obtained by severely lowering the facial ridge. The last group (4) is total tympanoplasty, in which the stapes is also missing and the columella effect cannot be obtained. They leave the round window in the mastoid cavity, turn the mucous membrane of the promontory down over the oval window, covering this with a graft to form a small closed air pocket connected with the Eustachian tube. Their results are very good in Group 1, diminishing to moderate improvements in Group 4.

Thorburn[33] (Blackpool, England) reported an interesting case of dislocation of the incus from the stapes which followed a severe head injury 14 years before the patient was seen. There had been bleeding from the ear for a few days but no vertigo and no infection. The hearing had been badly damaged at once and had remained stationary. Before operation the audiogram showed a loss of 40 db. by air and normal by bone conduction. At operation the dislocated incus was removed; the drum was mobilized posteriorly to bring it into contact with the stapes to achieve a columellar type repair as described by Wüllstein as Type 3. The resulting hearing improvement was excellent, being normal up to the 2,000 cycle range with a drop in the high tones.

(David Deweese described a similar traumatic dislocation of the stapes, diagnosed preoperatively at the 1958 meeting of the "Triological" Society, Western Section. It will be published in the Transactions.)

Cholesteatoma

Rüedi[34] (Zurich, Switzerland) presented before the International Congress of Otolaryngology at Washington, a very strong argument for the immigration theory of the formation of all types of cholesteatoma of the middle ear. He presented excellent microscopic sections of the epidermis of Shrapnell's membrane to show that "all healthy embryos, newborns, and infants show an increased growth in the sense of hyperkeratosis and acanthosis" in this area. "The special energy of growth so typical of the matrix of cholesteatoma may be derived from this section of epidermis—even without a primary perforation. Among the factors inducing growth—the irritation provided by a necrotizing or fistulizing otitis media contributes decisively in many cases."

After microscopic examination of hundreds of cases he found no evidence to support the metaplasia theory (in which the cuboidal epithelium is said to be transformed into stratified squamous epithelium from which cholesteatoma originates). In 40 cases of otitis media without perforation there was evidence of additional pro-

liferation of the epithelium of Shrapnell's membrane.

He concluded by advising early operation on all forms of cholesteatoma with as complete removal of the matrix as possible. He also advised a preventive antroatticotomy and removal of connective tissue layers in the epitympanum in cases of recurrent infantile acute otitis media.

(His illustrations are excellent, this is a paper based on a great deal of histological and clinical work, well worth careful study.)

Simpson[35] (Adelaide, Australia) reported a case of cholesteatoma of the petrous apex which was not suspected until he developed a fulminating fatal meningitis. It was not possible to decide whether the cholesteatoma developed from the ear or was of the rarer intracranial epidermoid variety. The patient was a 58-year-old gardener who had had chronic otitis for 48 years with facial paralysis for most of this time and facial neurolgia for 17 years. A few months before death he had an attack of vertigo diagnosed as Ménière's syndrome. Autopsy showed an enormous cholesteatoma with no evident connection with the ear. The author had seen the case only after the patient became unconscious. He advised that any Ménière's syndrome or slowly progressive facial paralysis be examined by x-ray for possible intrapetrous lesions.

Cawthorne[36] (London) at the Washington International Congress of Otolaryngology reported on a condition which he called perilabyrinthitis. He has seen about 15 patients in the past 12 years with severe disturbances of equilibrium, all of whom had been subjected to one or more mastoid operations. They all had very active responses to the fistula test and to caloric stimulation.

In none of the cases was there any active infection. Their gaits were so bizarre and symptoms so severe that most of them had been labeled as hysterical. One of these cases had had a fenestration operation for otosclerosis. She not only was almost disabled by the resulting giddiness but was unable to tolerate certain loud noises. She nearly fell off the chair when tested with a 2,000 tone to 80 db. but was not disturbed by loud tones at other frequencies.

These patients were all relieved of their symptoms by destruction of the labyrinth.

He concluded that they were suffering from a Tullio phenomenon, in which the fistula in the bony labyrinth allowed unaccustomed fluctuations to occur in the labyrinth because of a mobile stapes.

Petrositis

Rambo[37] (New York) and Petti (Yonkers) reported a case of the now unusual petrous apicitis occurring in a girl of 5 years. She had a bilateral otitis media with β-hemolytic streptococci in both. The right ear cleared but the left required a simple mastoidectomy 17 days after the onset. Two weeks later there began paralysis of the left lateral rectus muscle, and diagnosis of apicitis was made after x-ray showed involvement of the petrous tip.

Two attempts were required to reach the cavity in the petrous apex, the second being made along the carotid canal. Culture of the pus from the cavity showed the same organism, the β-hemolytic Streptococcus, but it was no longer sensitive to the previously indicated antibiotics. Recovery was complete, with disappearance of the paralysis of the lateral rectus muscle on the 10th postoperative day.

Intracranial Complications

Johnson and Smith[38] (Toronto) reported their method of differential diagnosis between intracranial and vestibular lesions. Strong emphasis is placed on close cooperation with the neurologist. The history is important; a story of distortion of sounds such as sensitivity to loud noises is taken as evidence of peripheral origin. They use the caloric tests described by Hallpike and also the rotation tests. The findings are frequently a great help to the neurologist.

(Those interested are referred also to the excellent article by Dr. Lyle Sellers, reported last year.)

Uihlein [39] (Mayo Clinic, Neurosurgical Dept.) reported the interesting case of a woman 58 years old who developed a huge subdural empyema covering the entire left cerebral hemisphere.

Intracranial symptoms developed after only five days of acute otitis media. The patient recovered after surgical removal by craniotomy. The mastoid was apparently not involved and was not operated on. The complication was apparently caused by thrombophlebitis of the cortical veins. The symptoms of intracranial pressure developed suddenly and were rapidly progressive, resembling a rapidly growing brain abscess.

Loomis [40] (Winona, Minn.) reported an interesting case of a 39-year-old man who developed suddenly fatal convulsive meningitis and bilateral lateral sinus thrombosis within two days of the onset of acute otitis media. He had been seen within a few hours of the onset; myringotomy was done, and erythromycin 250 mg. given every six hours. Within 24 hours meningitis developed, which was rapidly fatal in spite of large doses of penicillin. Autopsy, however, showed there was a chronic otitis with much old granulation tissue present which had given no symptoms before the acute onset.

Secretory Otitis Media

Bateman [41] (London) divided his 67 cases of secretory otitis media into three groups. His first group of 17 children under 15 years of age were almost all cured by removal of obstructing adenoids. His second group of five adults developed symptoms after flying, usually with respiratory infections. The third group of 41 patients had stuffy noses from many causes—hay fever, sinusitis, nasal polypii. Two of them had nasopharyngeal malignancies.

Many of his cases cleared up following inflation of the Eustachian tubes. He found that where paracentesis was necessary he was able to clear out the secretion most effectively by applying air pressure to the external canal with the Siegel scope. He then teaches his patients to autoinflate, partly to help prevent recurrences while flying. He mentioned the excellent description of the condition by James Hinton, written in 1874, stating that the illustrations were excellent, and that the treatment described was the same as we use today. In discussing the paper, Air Vice-Marshal E. D. D. Dickson reported a stubborn case of serous otitis media in which the secretion was very dark. After repeated evacuations a mastoidectomy was done and all cells were found to be filled with the same dark viscid secretion. The condition recurred after mastoidectomy.

Armstrong [42] (Charlotte, N. C.) treated 69 cases of chronic secretory otitis media by myringotomy and introduction of small plastic tubes for prolonged ventilation. During the two and one-half year period in which these patients were seen he also saw 947 cases with secretory otitis which cleared up without use of tubings. He obtained good results without recurrence in 60 ears and good results in 8 other cases after one or more recurrences. One case developed purulent otitis media after introduction of the tube.

He did not discuss etiology but expressed the opinion that the condition is apparently becoming much commoner and suggested that the use of antibiotics with resulting incomplete resolution might be a cause.

He suggested a test for serous otitis which he calls the binaural transmission test. He uses a diagnostic tube in each ear connected to each ear of the patient. He found that the patient's voice (or a tuning fork held on the patient's head, as for the Weber test) is heard much louder through the affected ear. This may be a valuable test where the usual fluid level is not present and when treating an apprehensive child. He uses a special tubing made by Becton, Dickinson and Co., called number

444 T. This tubing may be sterilized by autoclaving. He uses a piece 1 cm. in length cut with 45 degree bevel. He has not seen any permanent perforation even after use as long as three months.

(These excellent results should encourage us to try this method in chronic, resistant cases.)

Boyd [43] (New York) reported a case of resistant serous otitis treated by a new technique. After the condition had resisted the usual treatments a small plastic tube 1 mm. in diameter was left in the middle ear for 51 days. The tube, instead of being inserted through the drum, was inserted below the drum through an incision such as is made for stapes mobilization. The serous otitis was controlled during the 18 months that the patient was followed. Boyd thinks there is less possibility of a residual perforation and infection when the tube is inserted in this manner.

Persky [44] (Philadelphia) is convinced that serous otitis media is much commoner in the last 10 years and thinks that this may be due to incomplete treatment of acute otitis media by antibiotics during acute attacks. "The sterilization of an otitis media by antibiotics and residual fluid undrained by paracentesis sets the stage for organization of the inflammatory exudate and permanent impairment of hearing." He also lists as causes of serous otitis: allergies; hypertrophy of lymphoid tissue at the opening of Eustachian tubes, and new growths.

He advises that antibiotic treatment (penicillin is still his first choice) should be prolonged and assisted by paracentesis where necessary. He has seen little result following radiation therapy.

Evers [45] (Bend, Ore.) reported two cases of serous otitis caused by tumors of the nasopharynx. Both patients had treatments for several months with myringotomies before the tumors were recognized, even though the nasopharynx was examined on the first visit. He concluded that careful, repeated examination of the nasopharynx is necessary, even if general anesthesia is required.

External Otitis

Senturia [46] (St. Louis) reported a 30% incidence of the development of fungi and yeasts in ear canals treated with aqueous solutions of neomycin and hydrocortisone together with an ointment containing the same medications. Since the ointment alone did not cause otomycosis, he concluded that the continued wetting of the skin was responsible. He considers the use of topical corticosteroids as the most important advance in treatment of external otitis in recent years, but advises against overtreatment with the aqueous preparations. In 57 clean fenestration cases that were packed with neomycin-hydrocortisone suspension and ointment there was a 33% incidence of otomycosis. He stressed the phrase "control and sometimes cure" since control of symptoms was more frequent than permanent cures. His beautiful colored illustrations of the microscopic appearance of the skin of the ear canal are worth examination. He also reported, as have others, the high incidence of external otitis in the humid months of the year.

Perry,[47] instructor in dermatology at Wayne State University, has written an interesting monograph of 116 pages, "The Human Ear Canal." He covered the anatomy and physiology of the normal canal, and then describes the different types of external otitis media and their treatment. He found that rubbing or handling of the external ear would cause a spilling out of wax on the skin of the canal in the form of tiny, white, watery droplets, seen only with magnification. If this is collected by capillary pipettes and kept for a few days it would gradually darken in color. In many patients there was no secretion of wax at any time. Pain or the anticipation of pain would cause his subjects to "arrive at the laboratory with wet ear canals and, consequently, empty ceruminous gland reservoirs." Heat was not capable of emptying the ceruminous glands, while eccrine sweat glands respond vigorously to heat. Both the ceruminous glands and the apocrine

glands of the human axilla are "reservoir glands," and once the reservoir has been emptied a subsequent response is impossible until refilled. They are histologically identical.

In treatment he suggests that, in general, the more acute and inflammatory the process, the milder and more soothing the remedy should be. A weeping, crusted ear canal must be handled gently with wet dressings, such as Burow's solution diluted 1:20. In dry, chronic, and dry seborrheic dermatitis the ointments are to be used. Ointments and suspensions of hydrocortisone are most valuable, especially when combined with polymyxin, neomycin, and bacitracin.

(The otologist will profit by the dermatologist's point of view. This is an excellent monograph.)

Bihari [48] (London) treated 34 patients with chronic ear infections, using a solution of hydrocortisone, polymyxin and neomycin. Sixteen of these cases were external otitis, six were chronic otitis media and twelve were cases of infection of old mastoid cavities. Most of them had resisted treatment by various powders and solutions. There was no toxic or sensitizing reaction seen.

Thirteen cases were cured, nineteen were improved, and no effect was noted in two cases. He was convinced that the addition of hydrocortisone was very effective because the breaking of the vicious circle of irritation, scratching, and infection. A few patients would have relapses which required periodic treatment. Their method of treatment consisted of cleaning by dry mopping and instillation of 3 drops of the solution three times daily.

Perry [49] (Philadelphia), a dermatologist, urged that we approach the diagnosis of external otitis with the idea that a dermatitis of the external auditory canal can represent the first manifestation of a systemic disease. He particularly advises checking for skin lesions in other parts of the body. His excellent illustrations contain one in which a mild scaly lesion was diagnosed as a psoriasis when a typical lesion was found on the patient's knee. Another case was diagnosed as seborrheic dermatitis when the typical scaling and serous exudate was found behind the ear.

"All the usual causes of pruritis must be investigated, including diabetes, liver disease (even though jaundice is not evident), lymphoblastoma, and urticaria." He has been able to produce soggy maceration of the skin, "hot-weather ear of the tropics," by use of a rubber or plastic plug kept in the ear canal for two to four weeks. The canals became normal in two to four days after removal of the plugs. He does not discuss treatment but thinks that proper treatment will follow proper diagnosis. (The reviewer is reminded of a case of resistant dermatitis in a man of 40. After a few visits, when the usual treatment showed no improvement, he was advised to have an examination by an internist. Early diabetes was found.)

Dossetor [50] (Newmarket, England) reported on 18 months' observation of 300 cases of external otitis seen in troops in Aden during 1949 and 1950. He found the commonest organism to be Bacillus pyocyaneus. He found the new cases to double during the monsoon seasons when humidity was greatest. He obtained best results by a mixture of equal parts of acriflavine 1:500 and alcohol with the addition of 1.5% Phenoxetol. This was markedly superior to an aqueous mixture such as Burow's solution.

Fabricant [51] (Chicago) measured the pH factor in the ear canals of 11 patients with external otitis. They were all decidedly alkaline. He had previously reported measurements of normal ear canals as being on the acid side. These findings show the need of using medications which will restore the reaction to the normal acid physiological state.

Miscellaneous

Harbert and Sherk [52] (Philadelphia) reported the unusual case of spontaneous ex-

trusion of the incus during otitis media. The patient was a 2-year-old Negro boy who had pain and discharge from the ear one month before admission, subsiding after a few days. Within a week the patient complained of discomfort in the ear, and the mother removed an object from the canal which proved to be the incus. An enlarged photograph shows an almost normal incus. Examination a few months after onset of the otitis showed only a granulating healing perforation. Three months later the drum appeared normal, and there was not more than 10 db. loss when he was tested with the speech audiometer.

They found no recent reports of a spontaneous extrusion of an ossicle but found that in several old texts the possibility was mentioned.

Huntley and Dees [53] (pediatricians at Duke University School of Medicine) reported five cases of children, all of whom died of a combination of purulent otitis media, eczema, and thrombocytopenic purpura. There was a definite hereditary family tendency found in three of the cases, and all patients were males. It was thought that these cases may represent a definite syndrome and that similar cases should be reported.

Splenectomy was done on four of the five cases for treatment of the purpura. There was improvement, but it was noted that their resistance to infection was definitely impaired after operation. The last case seen was examined for γ-globulin and found to have normal level. The earlier cases did not have this examination.

Hall [54] (Oslo, Norway) reports the unusual case of a woman of 49 who developed deafness in one ear without pain. Examination showed a blue retracted drum. At first she would be helped by paracentesis and removal of a brownish secretion by suction. After several months a simple mastoidectomy was done, and many bleeding granulations were found in a rather cellular mastoid. The hearing became normal after operation, and the blue drum became normal in color. He was able to find only 12 cases reported previously.

Since this paper was written Birell has reported 21 cases of black cellular cholesteatosis in childhood. Tumarkin, in discussing these darkly discolored cellular mastoids states: "We may ask what is this sanguineous response which seems to occur specifically in the cellular mastoid. We stumbled on a possible clue in considering the reservoir function of the accessory air cells. We have seen that when Eustachian obstruction occurs, the effect on a cellular mastoid can be even more disastrous than on the acellular bone. The resulting vacuum is only too likely to set up hemorrhage—absorption would be slow—with recurring slight hemorrhage from the engorged granulations—producing the familiar picture of the blue eardrum."

Zielinski and Oren [55] (Tel-Aviv) reported the unusual case of complete bilateral deafness and destruction of labyrinthine function due to mumps. The deafness and loss of balance preceded the parotitis by 21 days. The patient was a boy 3½ years of age. The child first became ill with a mild fever, and the first diagnostic symptoms of the labyrinthitis came a week later when the child began to stagger and fall. A few days later he became deaf. Repeated prolonged irrigations of both ears with ice water produced no reaction. Lumbar puncture and x-rays of the skull were negative. Three weeks after deafness the parotid glands began to swell, and the diagnosis of mumps was confirmed by measurement of the blood diastase, which rose to 190 units, and the urinary diastase, which rose from 512 units to 1,000 units. The boy's speech, which had been good, rapidly deteriorated until 50 days after the onset of illness his speech was incomprehensible. His general condition remained excellent, and his gait improved. Corticotropin (ACTH) was given daily for 10 days without result.

960 E. Green St.

REFERENCES

1. Tumarkin, A.: On the Nature and Vicissitudes of the Accessory Air Spaces of the Middle Ear, J. Laryng. & Otol. 71:65-99 (Feb.); 137-161 (March); 211-248 (April) 1957.

2. Diamant, M.: Anatomical Etiological Factors in Chronic Middle Ear Discharge, Ann. Otol. Rhin. & Laryng. 66:380-389 (June) 1957.

3. Schuknecht, H. F., and Trupiano, S.: Some Interesting Middle Ear Problems, Tr. Am. Laryng. Rhin. & Otol. Soc., pp. 130-148, 1957.

4. Friedmann, I.: The Pathology of Otitis Media with Particular Reference to Bone Changes, J. Laryng. & Otol. 71:313-320 (May) 1957.

5. Parr, E. Q., and Hallberg, O. E.: Fever of Obscure Origin, A. M. A. Arch. Otolaryng. 65:300-303 (March) 1957.

6. Rosenwasser, H., and Adelman, N.: Otitic Complications, A. M. A. Arch. Otolaryng. 65: 225-234 (March) 1957.

7. Davison, F. W.: Effective Antibiotic Therapy, A. M. A. Arch. Otolaryng. 66:714-718 (Dec.) 1957.

8. Acute Otitis Media in General Practice, Report of a Survey by the Medical Research Council's Working-Party for Research in General Practice, Lancet 2:510-514 (Sept. 14) 1957.

9. Sanderson, B. A.: Conservative Management of Chronic Otitis Media, A. M. A. Arch. Otolaryng. 66:170-171 (Aug.) 1957.

10. Goldstein, L. J.: Bacterial Resistance to Antibiotics in Chronic Otitis Media, A. M. A. Arch. Otolaryng. 65:454-455 (May) 1957.

11. Fritz, M. H.: Mastoiditis in Alaska, Northwest Med. 56:563-577 (May) 1957.

12. Moore, J. A.: Endaural Modified Radical Mastoidectomy, Tr. Am. Laryng. Rhin. & Otol. Soc., pp. 408-432, 1957.

13. Ohrt, H. H.: On Recurrence in Cavities After Radical Mastoidectomy with Special Reference to the Complications Which May Arise, Acta oto-laryng. 47:346-352 (April) 1957.

14. Lee, J. H. A.: Chronic Otitis in a Sample of Young Men, J. Laryng. & Otol. 71:398-404 (June) 1957.

15. Aschan, G., and Nohrman, B.: X-Ray Treatment of Otosalpingitis, Acta oto-laryng. 47:1-9 (Jan.) 1957.

16. Davison, F. W.: Atticomastoidectomy, Laryngoscope 67:191-200 (March) 1957.

17. Rohan, R. F.: Artificial Tympanic Membranes, Old and New, J. Laryng. & Otol. 71:605-615 (Sept.) 1957.

18. Turner, J. L., and Dobson, H. L.: Use of Gamma Globulin in the Chronically Ill Child, Laryngoscope 67:1286-1296 (Dec.) 1957.

19. Davison, F. W.: Atticomastoidectomy, Tr. Am. Soc. Laryng. Rhinol. & Otol. Soc., pp. 34-45, 1957.

20. Moore, J. A.: Endaural Modified Radical Mastoidectomy, Laryngoscope 57:521-543 (June) 1957.

21. Goodhill, V.: Otosurgical Developments and the Hard of Hearing Child, Tr. Am. Acad. Ophth. 61:711-722 (Nov.-Dec.) 1957.

22. Zöllner, F.: The Meatoantrotomy—A Special Approach for Plastic Procedures, Ann. Otol. Rhin. & Laryng. 66:547-552 (June) 1957.

23. Zöllner, F.: The Prognosis of the Operative Improvement of Hearing in Chronic Middle Ear Infections, Ann. Otol. Rhin. & Laryng. 66:907-917 (Dec.) 1957.

24. Rambo, J. H. T.: A New Operation to Restore Hearing in Conductive Deafness of Chronic Suppurative Origin, A. M. A. Arch. Otolaryng. 66:525-532 (Nov.) 1957.

25. Bell, H. L.: Technique of Tympanoplasty, A. M. A. Arch. Otolaryng. 66:554-571 (Nov.) 1957.

26. Pietrantoni, L., and Bocca, E.: Myringoplasty, Ann. Otol. Rhin. & Laryng. 66:1160-1172 (Dec.) 1957.

27. Juers, A. L.: Principles of Hearing Conservation in Surgery for Chronic Ear Disease, Ann. Otol. Rhin. & Laryng. 66:710-715 (Sept.) 1957.

28. Pick, E. I.: Surgical Repair of the Sound Conduction Mechanisms of the Middle Ear, Ann. Otol. Rhin. & Laryng. 66:1044-1063 (Dec.) 1957.

29. Hall, A., and Rytzner, C.: Stapedectomy and Autotransplantation of Ossicles, Acta oto-laryng. 47:318-324 (April) 1957.

30. Miodonski, J.: Columellization and Mobilization of the Middle Ear, Acta oto-laryng. 47:64-72 (Jan.) 1957.

31. Beales, P. H.: Early Experiences of the Plastic Surgery of the Sound Conducting Apparatus with Report of 57 Consecutive Cases, J. Laryng. & Otol. 71:162-174 (March) 1957; 297-312 (May) 1957.

32. Ormerod, F. C., and McLay, K.: Some Results of Plastic Operation on the Sound Conduction Apparatus, J. Laryng. & Otol. 71:427-433 (July) 1957.

33. Thorburn, I. B.: Post-Traumatic Conduction Deafness, J. Laryng. & Otol. 71:542-545 (Aug.) 1957.

34. Rüedi, L.: Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone, Ann. Otol. Rhin. & Laryng. 66: 283-305 (June) 1957.

35. Simpson, D. A.: "Cholesteatoma" of the Petrous Apex, J. Laryng. & Otol. 71:548-553 (Aug.) 1957.

36. Cawthorne, T.: Perilabyrinthitis, Laryngoscope 67:1223-1236 (Dec.) 1957.

37. Rambo, J. H. T., and Petti, G. H.: Petrous Apicitis, A. M. A. Arch. Otolaryng. 65:523-524 (May) 1957.

38. Johnson, W. H., and Smith, J. B.: Differentiation of Intracranial and Peripheral Vestibular Lesions, Tr. Am. Laryng. Rhin. & Otol. Soc., pp. 438-449, 1957.

39. Uihlein, A.: Acute Subdural Empyema of Otitic Origin, Minnesota Med. 40:31-34 (Jan.) 1957.

40. Loomis, G. L.: Report of a Death Following Acute Otitis Media, Ann. Otol. Rhin. & Laryng. 66:499-502 (June) 1957.

41. Bateman, G. H.: Secretory Otitis Media, J. Laryng. & Otol. 71:261-270 (April) 1957.

42. Armstrong, B. W.: Chronic Secretory Otitis Media, South. M. J. 50:540-546 (April) 1957.

43. Boyd, G. G.: Otitis Media, A. M. A. Arch. Otolaryng. 66:489-492 (Oct.) 1957.

44. Persky, A. H.: Serous Otitis Media, Am. Practitioner 8:580-582 (April) 1957.

45. Evers, L. B.: Otitis Media with Effusion Associated with Nasopharyngeal Tumors, Northwest Med. 56:582-585 (May) 1957.

46. Senturia, B. H.: What's New in the Treatment of Otitis Externa, the Otologist's Viewpoint, Tr. Am. Acad. Ophth. 61:347-359 (May-June) 1957.

47. Perry, E. T.: The Human Ear Canal, Springfield, Ill., Charles C Thomas, Publisher, 1957.

48. Bihari, J.: Chronic Ear Infections: Local Treatment with Hydrocortisone, Neomycin, and Polymixin Combined, Brit. M. J. 2:623-625 (Sept. 14) 1957.

49. Perry, E. T.: A Practical Approach to External Otitis, J. A. M. A. 163:161-164 (Jan. 19) 1957.

50. Dosseter, A. E.: A Report on Otitis Externa, J. Laryng. & Otol. 71:271-275 (April) 1957.

51. Fabricant, N. D.: The pH Factor in Treatment of Otitis Externa, A. M. A. Arch. Otolaryng. 65:11-12 (Jan.) 1957.

52. Harbert, F., and Sherk, H. H.: An Unusual Complication of Otitis Media, A. M. A. Arch. Otolaryng. 65:45-46 (Jan.) 1957.

53. Huntley, C. C., and Dees, S. C.: Eczema Associated with Thrombocytopenic Purpura and Purulent Otitis Media, Pediatrics 19:351-361 (March) 1957.

54. Hall, J. G.: Blue Eardrum, Acta oto-laryng. 47:245-250 (March) 1957.

55. Zielinski, B. Z. D., and Oren, M.: A Case of Mumps Labyrinthitis, J. Laryng & Otol. 71: 853-855 (Dec.) 1957.

Abstracts from Current Literature

Ear

EUSTACHIAN TUBE BIOPSY CANNULA. M. K. MUFTIC, Eye, Ear, Nose & Throat Month. 36: 735 (Dec.) 1957.

A simple routine procedure is described which permits a biopsy of the Eustachian tube orifice and of Rosenmüller's fossa. The instrument utilized is a modified Eustachian tube catheter with the end-piece designed after the fashion of an endometrial biopsy cannula-curette. The piece of tissue for biopsy engaged in the end of the cannula is held in position by means of strong suction, exerted through the Eustachian tube biopsy cannula.

JENNES, Waterbury, Conn.

IDENTIFICATION OF HEARING LOSS BY PARENTS, TEACHERS AND AUDIOMETERS. F. KODMAN JR., P. E. SCHNECK, K. G. STOCKDELL, W. V. THOMPSON, AND C. SPIES JR., Eye, Ear, Nose & Throat Month. 37:103 (Feb.) 1958.

The hearing of 194 elementary school children was tested by individual pure-tone audiometry. In addition the classroom teachers were requested to evaluate the same pupils as having either normal hearing or a mild to severe hearing loss, and the parents of the children tested were asked to perform a similar evaluation.

The incidence of hearing loss by audiometric examination was 28.10% for the 15-25 db. group, and 13.10% for the 30 db. loss group. Of the evaluations made by the teachers there were 71 incorrect judgments, and the parents were incorrect in 65 cases. Whereas it is felt that parents and teachers did better than chance in detecting pupils with mild to moderate hearing losses when referrals were not considered, both parents and teachers were making incorrect judgment in 4 out of 10 cases.

It is to be concluded that the audiometer is far superior to either the parent or the teacher as a detector of hearing loss. Annual hearing surveys followed by periodic medical examination are necessary to insure the well-being of our school children.

JENNES, Waterbury, Conn.

FAVORABLE RESPONSE OF BELL'S PALSY TO PREDNISONE (METICORTEN). N. D. FABRICANT, Eye, Ear, Nose & Throat Month. 37:116 (Feb.) 1958.

Two patients with Bell's palsy were treated with prednisone with excellent therapeutic results. Dosage consisted of six 5 mg. tablets for four days, with reduction of one tablet every fourth day until one tablet was prescribed daily. In the first patient reported, some improvement in the facial palsy was detected within five days, considerable improvement within two weeks, and complete clearing within three weeks. In the second patient improvement first appeared on the seventh day and there was pronounced improvement at the end of two weeks. During the third week, however, because of pain from a concomitant duodenal ulcer, the prednisone therapy was reduced at an accelerated rate. Almost complete recovery was evident after three weeks of treatment, and the pain due to the duodenal ulcer disappeared entirely.

JENNES, Waterbury, Conn.

THE CONTROL OF ITCHING OF THE EXTERNAL EAR WITH SANDOSTENE. WALLACE RUBIN and JACK R. ANDERSON, Laryngoscope 68:189 (Feb.) 1958.

Sandostene affects allergic processes. Its actions which are antipermeability, anticholinergic, and antihistamine, should make it effective against pruritis. It is taken by mouth, 50 mg. or more four times a day. Approximately 90% of 147 patients experienced relief. Although there were no controls in Rubin and Anderson's experience, about 30% of patients were relieved by other methods.

HITSCHLER, Philadelphia.

BASIC TECHNIQUES IN MYRINGOPLASTY AND TYMPANOPLASTY. FRED R. GUILFORD and WILLIAM K. WRIGHT, Laryngoscope 68:825 (May) 1958.

For a successful tympanoplasty certain conditions must be met. They are a dry ear, good cochlear reserve, good mucous membrane lining of the middle ear, and a functioning Eustachian

tube. It is well, in addition to the usual hearing tests, to place a patch on the perforation and then test, also to test with the acoustic probe. Both these aid in determining the condition of the ossicular chain.

Because of the need for blood vessels three-quarters or full-thickness graft is to be preferred for myringoplasty. The ear should be dry for 30 days preoperatively and all precautions taken to avoid infection at the time of the operation and postoperatively.

The exact technique of the procedure is described. It essentially consists of placing a three-quarters thickness graft taken from the mastoid area upon the denuded drum and canal wall, after both an anterior and a posterior flap of the skin of the canal wall have been made and laid back. Some canal bone is removed for better visualization.

Description of the other types of tympanoplasty follows. The Wullstein Type IV is divided into two parts, one with just erosion of the long process of the malleus, and the other with erosion of the long process of the incus in addition. In the absence of cholesteatoma, the latter can be treated surgically as a Type II, with certain advantages.

Types III, IV, and V are also discussed. These are similar to those described by Wullstein.

HITSCHLER, Philadelphia.

EARLY EXPERIENCE WITH TYMPANOPLASTY (WULLSTEIN TECHNIQUE). MILES L. LEWIS, Laryngoscope 68:900 (May) 1958.

This is a review of 16 cases of tympanoplasty. Any patient with chronic otitis media or mastoiditis is a candidate for tympanoplasty, provided there is good cochlear function and a patent Eustachian tube.

The technique is described. It is similar to that of Wullstein. It is noted that the pinning back of the ear due to the taking of the graft is not permanent. The ear resumes its normal position in several months.

The 14 Type III and IV tympanoplasties showed an average each of 16 and 14 db. respectively. Of two Type I cases, one resulted in normal hearing and the other in no gain.

HITSCHLER, Philadelphia.

OTOSCLEROSIS: A TWENTY-YEAR RETROSPECT. J. SIMSON HALL, Proc. Roy. Soc. Med. 51:57 (Jan.) 1958.

This is the President's address before the Section of Otology, Nov. 1, 1957. His interest was aroused in May, 1930, when he heard Prof. Maurice Sourdilles, of Nantes, France, expound his work on otosclerosis. The recommended surgical technique was taken up by Holmgren, but it was not well popularized in England until chemotherapy came into vogue and Lempert of New York City described his researches and the results after surgery. Mr. Hall has studied the aural pathology carefully and has concluded that "there is a sudden, probably inflammatory condition of the bone in which osteoclasis plays a large part." The reaction to irritation, whatever it is, causes a hyperostosis in the temporal bone which may be compared to that found in Paget's disease, sometimes "called a localized form of otosclerosis. . . . In fragilitas ossium the onset of deafness occurs with the healing of the bone, suggesting again that healing of bone and deafness are concurrent." On his first series of 50 patients, Mr. Hall used local anesthesia but became dissatisfied with it. At present he is using Thiopentone with or without a relaxant. After intubation, gas-oxygen is the chief agent. Blood pressure is lowered to from 50 to 80 mm. Hg. Induced hypotension is a calculated risk, but results have shown that it is justifiable. A thousand consecutive patients have been operated upon and classified into groups; from 65% to 70% to 90% of results were regarded as successful.

As for operative technique, "the whole crux of the matter rests on making a satisfactory opening into the labyrinth in such a way that the opening will remain patent." However, a discharging cavity has remained in some 13%. It seems to be "as great as before we had chemotherapy." . . . Strangely enough we are not informed as to the chemotherapy in use by the author, but the inference is that it is used. Skin grafting is carried out, with cauterization of exuberant granulations by trichloracetic acid where indicated. . . .

Mobilization "has come to stay," has very definite usefulness. "I do not now perform the fenestration operation until the effects of mobilization are known and have been tried. I consider that the one operation is complementary to the other." The author states that 8% of patients have had bilateral operations. "The speech audiograms make it clear that there is a very real gain in binaural hearing after operating upon the second ear." However, the technique used is subject to change as often as every six months. This seems to mean that perfection has

not yet been attained, which may be taken to mean that more and better results are to be expected.

At the Dec. 6, 1957, conclave, the discussion on stapes mobilization was opened by Dr. Samuel Rosen, of New York, on "Stapes Surgery For Otosclerotic Deafness," followed by papers from Mr. Terence Cawthorne and Mr. R. S. Venters. Others also took part. These writings will appear in the *Journal of Laryngology.*

I. W. VOORHEES, New York.

Larynx

OCCULT CARCINOMA OF THE ESOPHAGUS. DAVID B. MONHEIT and MORRIS SLOBODKIN, New York J. Med. 58:2506 (Aug. 1) 1958.

According to the Proceedings of the Third National Conference, the survival rate of cancer of the esophagus is about 2%—the lowest of any series studied. Reason: Early symptoms are few and nonspecific. Dysphagia may be the first, but it comes late. A slight discomfort behind the sternum, gas eructations, "heartburn," fullness, a cough which is not significant may be all. Perforation, mediastinitis, empyema, fatal hemorrhage may be "quite early." Metastases to adjacent organs and tissues are found in one-third of autopsies. Four cases are here reported in detail, with x-rays, etc., to illustrate the hidden character of this disease. None of the patients complained of dysphagia at any time." But pain in the chest was a common symptom.

ABSTRACTER'S COMMENT: Direct tube examination is almost certain to be called for when it is too late for surgery. The most one can do is to take a section for biopsy. Even so, the tissue may seem to be merely "inflammatory," which to me is entirely unsatisfactory and may be highly misleading. It must be admitted at this stage of our knowledge that we have no means of making an early diagnosis of cancer of the esophagus save through the esophagoscope. And nobody is going to submit to this discomforting procedure because of the "minor symptoms." Unfortunate, but that is the way it is!

I. W. VOORHEES, New York.

Nose

HEALING OF NASAL FRACTURES—A ROENTGEN EVALUATION. R. J. CAPAROSA and A. R. ZSVATSKY, Eye, Ear, Nose & Throat Month. 37:36 (Jan.) 1958.

A small series of nasal fractures, both treated and untreated, was studied by serial roentgenograms. A persistent fracture line may be noted on x-ray examination for years following nasal trauma. Complete healing as demonstrated by x-ray examination occurs in those patients in whom reduction was satisfactory, indicating that healing is directly related to the proximity of the fractured fragments. In cases of recent trauma to the nose, the presence of a fracture line by x-ray may have little significance in the abscence of the physical signs of a recent fracture. The deformity present may be due to an old injury. This is important not only from a medicolegal point of view but also in terms of surgical correction. Simple reduction would be indicated if the demonstrated fracture line was due to recent injury, whereas more radical surgery would be required if the deformity were due to an old injury.

ABSTRACTER'S COMMENT: This brief article is timely; it provokes thought on the management of nasal fractures, a subject of ever-increasing importance to the otolaryngologist. Exposure to rhinoplastic techniques enables the otolaryngologic surgeon to approach nasal fractures with an experience gained from many surgically induced fractures. Because roentgen evaluation of nasal fractures is so frequently misleading, the rhinologist learns with experience to depend on the clinical evidence afforded by a pains-taking examination. Where doubt exists as to whether the nasal deformity is recent or old, or a combination of both, or when the injury is extensive, those instruments required for an open reduction should be available at the operating table. If the fragments do not become realigned readily by the technique of closed reduction, an open reduction should be performed.

If the term "more extensive" were substituted for "more radical" in the description of the type of surgery required when the deformity is due to an old injury, it would remove a psychological barrier from the mind of the surgeon as well as from the minds of the referring physician and the patient. An open reduction constitutes the simple approach to new nasal fractures superimposed upon preexisting nasal deformities.

JENNES, Waterbury, Conn.

NASOPHARYNGEAL IRRADIATION. D. A. DOLOWITZ, Eye, Ear, Nose & Throat Month. 37:39 (Jan.) 1958.

Large masses of lymphoid tissue in the nasopharynx are best removed by surgical means. Irradiation should be reserved for the shrinkage of selected nodules of lymphoid tissue. Such irradiation is best performed with radium rather than by x-ray. The safety of such radiation is well illustrated by its use at The Johns Hopkins Hospital since 1924 without a single case of burn, slough, or carcinoma in either patients or staff members.

Conductive disease of the middle ear and of the Eustachian tube, which is secondary to obstruction, will be helped by such therapy. Radiation is useful as a supplement to antibiotics in the treatment of chronic infection in the nasopharyngeal lymphoid tissue.

JENNES, Waterbury, Conn.

TREATMENT OF RAGWEED HAY FEVER WITH POWDERED HYDROCORTISONE APPLIED INTRANASALLY. C. F. LAKE, G. B. LOGAN, and G. A. PETERS, Eye, Ear, Nose & Throat Month. 37:176 (March) 1958.

Eighteen patients, including three children, comprised the series studied in this report. The children received daily dosage of 5 mg. of hydrocortisone powder and one received 7.5. mg. Adults received a daily dose of 15 mg. The total daily medication was used in divided doses, three to four times daily, in an inhaler type of powder dispenser. Treatment was continued from Aug. 21 to Sept. 27, covering a period of over five weeks. During this time the ragweed pollen count varied from a low of 0 granules to a high of 637 granules per cubic yard for 24 hours. The count did not fall below 32 granules per cubic yard for 24 hours until the third week of September.

The results were classified on a basis of excellent, good, fair, and poor. Of the 18 patients, 6 results were classified as excellent, 8 as good, which meant that their condition was improved to the point that they had only minimal residual symptoms, and 3 were considered to have only a fair result as their symptoms were relieved approximately 50%. One patient was classified as having a poor result, in that after five days of treatment she felt worse than before the start of treatment. The improvement in respect to symptoms and objective findings began in three to five days after the use of the hydrocortisone powder was started.

All patients treated had ragweed hay fever which was not responding to hyposensitization, use of antihistamine drugs, or both. Three patients are described as having had other treatment in addition to the powdered hydrocortisone during the test period.

The authors carefully point out that this is not a controlled study, as individual variations and severity of reactions to pollen and individual variations in exposure to pollen make control studies of treatment of hay fever patients of dubious value. The authors do, however, feel that they can conclude that intranasal insufflation of hydrocortisone offers another effective and easily administered form of therapy for the patient with seasonal allergic rhinitis not controlled by conventional means. Such treatment is not accompanied by an increased incidence of secondary bacterial infection, and it does not cause symptoms of hypercortisonism. In only one of the 18 patients was there the possibility that the use of the hydrocortisone powder had caused local irritation.

JENNES, Waterbury, Conn.

TREATMENT OF ALLERGIC RHINITIS. L. A. PACKARD, Eye, Ear, Nose & Throat Month. 37:257 (April) 1958.

Three hundred eighty-two patients with seasonal and perennial allergic rhinitis were treated with a nasal spray containing cortisone and an antihistaminic agent in combination with the oral administration of repeat action tablets of the same antihistaminic agent. The nasal spray used in this series was Corticloron, which contains 0.5% cortisone acetate and 0.3% Chlorprophenpyridamine (Chlor-Trimeton) gluconate in an isotonic vehicle. The oral medication consisted of Chlor-Trimeton maleate in the form of 8 mg. and 12 mg. repeat action tablets. Seven patients with nasal polyps were also treated with the same regimen.

The author reports that there were no treatment failures and that most of the patients obtained a fair degree of symptomatic relief. Of the 270 patients who received 8 mg. dosage of Chlor-Trimeton, the majority obtained a major degree of symptomatic relief. Of the 112 patients receiving the 12 mg. dosage of Chlor-Trimeton, complete relief was achieved in 25% sufficient relief to remain comfortable and without complaint was achieved in 50%, whereas the remaining 25% obtained symptomatic relief but their hay fever recurred when they inter-

rupted treatment. Of the seven patients with nasal polypi, which were described as ranging in size from small to the large obstructive type, the polyps disappeared from all patients in from one to four weeks of treatment.

Additional treatment consisted of the use of Meticorten administered orally where indicated and in the elimination of certain foods in those cases of allergic rhinitis associated with food allergy. Pollen desensitization was apparently not utilized.

In a footnote to the article it is commented that an additional 250 patients with seasonal or perennial allergic rhinitis have been treated in the manner outlined with the exception that Metreton Nasal Spray has been used in the place of Corticloron. This contains the corticoid prednisolone instead of cortisone acetate. The results are equal to or better than those achieved with Corticloron.

JENNES, Waterbury, Conn.

Pharynx

MIRROR VISUALIZATION TECHNIQUE OF NASOPHARYNGEAL SURGERY. M. GROSSMAN, Eye, Ear, Nose & Throat Month. 37:111 (Feb.) 1958.

The author describes his technique for performing adenoidectomy, which consists primarily in the use of the Davis-Crowe technique with the patient supine, the head hyperextended, and with the surgeon sitting at the head of the table. The Davis-Crowe mouth gag with attached ether tube hangs from the ether screen, allowing a change in position of the table without the necessity for changing the position of the mouth gag. A urethral catheter with holes cut for suction is introduced through the nose into the nasopharynx and then out through the mouth; it is then clamped with a heavy hemostat and is utilized both as a retractor and for the purpose of continuous suction of the operative area. The nasopharynx is visualized with the aid of a laryngeal mirror, coated with pHiso/Hex to prevent fogging, for the purpose of evaluation of the pathology and as a guide to the surgery.

JENNES, Waterbury, Conn.

NEWER CONCEPTS CONCERNING THE PREVENTIVE INFLUENCE OF TONSILLO-ADENOIDECTOMY ON THE COMMON COLD IN CHILDREN. A. R. HOLLANDER, Eye, Ear, Nose & Throat Month. 37:327 (May) 1958.

The existing pathologic state of the tonsils and adenoids should serve as the primary factor governing the indication for their elimination. Adequate preoperative diagnostic studies are absolutely essential if the common cold is to be differentiated correctly from repeated attacks of nasal sinusitis or persistent allergic episodes. There can be no causal relationship between the pharyngeal lymphoid structures and the common cold or other respiratory tract disease unless the existing tonsillar disease falls in the category of focal chronic tonsillitis. Because adenoids exist as one of the primary sources of headcolds in children, early adenoidectomy is the indicated procedure to reduce their incidence. Adequate preoperative diagnostic studies are essential if the common cold is to be differentiated correctly from repeated attacks of nasal sinusitis or persistent allergic episodes. There is, however, no single remedy which can produce the desired results in all patients. Multiple factors must therefore be considered in reducing the susceptibility to colds.

In his discussion of this paper, H. Grant Preston further clarifies this subject. The child whose tonsils and adenoids are being evaluated for removal should be considered as a complete entity. The pediatric indications for this surgery include anorexia, asthenia, inanition, and recurrent upper respiratory infections, which, if accompanied by a history of recurrent attacks of tonsillitis or recurrent otitis media, should call for operation. The indications for removal are clear if these pediatric indications are accompanied by a combination of certain surgical indications such as chronically inflamed pillars, infectious material deep in the tonsil crypts, or persistent cervical adenopathy. Preston stresses the necessity of the persistence of signs to constitute a true indication. The presence of beta hemolytic Streptococcus in the tonsils of convalescent or suspected rheumatic fever cases is another pediatric indication for surgery. The incidence of recurrent colds as the major pediatric problem, when accompanied by a combination of the above listed evidences of chronic adenotonsillar infections, can be expected to be reduced materially by the removal of the tonsils and adenoids.

JENNES, Waterbury, Conn.

CANCER ALERTS: MALIGNANT DISEASE OF FACIAL SKIN, LIPS AND OROPHARYNX. JEROME P. WEBSTER, New York J. Med. 58:2419 (July 15) 1958.

Webster is a general plastic surgeon at the Presbyterian Hospital and the Columbia University College of Physicians and Surgeons. This is No. IX in a series of cancer alerts. He advises every physician to examine the nature of every blemish, wart, mole, etc., on the faces of patients who come in for whatever purpose. This is of especial significance to all ENT specialists who may be more concerned with the intranasal and intraoral conditions than with externals. Any lesion which is "open" or heals poorly or slowly is under suspicion. The diagnosis should be made at first hand by whatever means, or, in case of doubt, some specialist in authority should have the referral. Most of these lesions in the skin can be cured promptly under proper management if attacked *early*. It is surprising how late many come in—sometimes too late. Capable pathologists are to be found in every Grade A medical school. Their word may well be considered final. Then comes therapeusis. No one should meddle with such lesions, surgically or otherwise, without expert advice and help, sharing the responsibility by consultation. While radiotherapy is more often advised than surgery, there is no vital reason why both should not be employed according to the problem at hand. As for surgery, it should be "radical" enough to eliminate the initial lesion, even if an "ugly result" is obtained. If we save a life thereby, plastic surgery can be resorted to at any later time. This is an important contribution.

I. W. VOORHEES, New York.

Miscellaneous

MOUTH BREATHING IN CHILDREN: AN EDITORIAL. A. R. HOLLANDER, Ear, Eye, Nose & Throat Month. 37:189 (March) 1958.

Adenoidal obstruction is not invariably the cause of mouth breathing in children. Despite the impressions to the contrary left by most medical writings, other pathologic processes must be considered as causes for such mouth breathing. These include deviated nasal septum, hypertrophic turbinates, infected accessory sinuses, nasal allergies, as well as that entity known as habitual mouth breathing. The facial expression termed adenoid facies is therefore not always attributable to massive adenoids. This expression may not only be caused by these other entities but may be accounted for by a hereditary relationship as well. The consequences of persistent mouth breathing in children are serious and affect the entire facial skeleton. The facial expression becomes dull and drawn.

The treatment of mouth breathing will obviously depend upon the cause. Surgical removal of hypertrophied adenoids and intranasal surgery for gross obstructive processes must be performed if the indications are absolute. A complete allergic study is in order if allergy is the underlying cause of the mouth breathing. In the absence of intranasal anatomic irregularities, an allergy is commonly the cause of mouth breathing, and explains why adenoidectomy frequently fails to cure mouth breathing. This is an important subject and should not be minimized, since mouth breathing is not only an annoying condition but can lead to secondary pathologic changes in the face, oral cavity, and in other structures of the respiratory tract.

JENNES, Waterbury, Conn.

PHOTOGRAPHY IN OTOLARYNGOLOGY. N. D. FABRICANT, Eye, Ear, Nose & Throat Month. 37: 338 (May) 1958.

Photography has assumed a very important role in otolaryngology. It serves as a valuable aid in medical teaching and research. It may be used to improve the morale of patients experiencing slow recovery from their disabilities. In cases involving compensation insurance, photographic evidence presents an effective story in simple and concise terms more readily understood than the most carefully explained medical terminology. Photographs aid in planning corrective surgery and serve as pre- and postoperative records. In cases of patients who have submitted to plastic operations of the nose or face who may fail to recall their former appearance or may have expected greater improvement than was possible to obtain through surgical means, a preoperative photographic record of the patient's face becomes an invaluable document.

Time, patience, and money are requisites for some of the subdivisions of photographic activity. These include infrared photography, color photography, motion pictures, and photomicrography. Progress in motion picture photography has been evident in the fields of laryngeal, aural, nasopharyngeal, and endoscopic photography, due to the development of superior motion picture cameras, and through quality of film and a better understanding of illumination. The Biological Photographic Association was formed more than a quarter of a century ago by a

small group of clinical photographers and the Journal of this organization can be the source of much information to physicians interested in motion picture or still photography.

Fabricant concludes that good medical photographs are a mark of distinction and paraphrases a well-known comment, "By your photographs t..ey will know you."

ABSTRACTER'S COMMENT: The otolaryngologist's interest in photography is evidenced by the frequency with which photographs appear as illustrations in our otolaryngological medical literature. It is, however, noteworthy to find an article written about photography, and especially when there is such a strong emphasis on quality. Methods of photography must be standardized and must be capable of being repeated without change, in order to obtain an acceptable comparison between pictures taken before treatment or during the course of or after treatment. "Before" and "after" sets of pictures must have objectivity if they are to have value. Differences in the methods of posing, in the choice of lens, and in the methods of developing film may introduce a factor of subjectivity into the photographic evidence. Perhaps photographs, like statistics, may not lie, but when objectivity is removed, albeit not wilfully, scientific objectivity becomes the loser.

JENNES, Waterbury, Conn.

FIXATION OF FRACTURES IN THE MIDDLE THIRD OF THE FACE, WITH KIRSCHNER WIRES. HERBERT H. HARRIS, Laryngoscope 68:95 (Feb.) 1958.

Because of traffic accidents, head and face fractures are becoming more frequent; consequently their management is more important. Severer injuries to other parts of the body must not be overlooked, and often have to be attended to first. Hemorrhage anywhere must of course be controlled at once. Nasal packing usually suffices for injuries in that area. If possible repair facial lacerations early in order to obtain a good cosmetic result.

Diagnosis is made by inspection, palpation, and x-ray. Stereo plates in the Waters position are to be preferred over flat plates.

Several methods of reduction of fractures of the face are available, but Harris pefers the use of the Kirschner wires for most of those that need fixation. Various kinds of fractures are listed and the method of using the wire described in detail together with illustrations.

Less time is required in the use of the Kirschner wire. There is little scarring and traction can be applied. No accidents or infections have so far been encountered in their use.

HITSCHLER, Philadelphia

THE TREATMENT OF BRONCHIAL ASTHMA IN CHILDREN. VINCENT FONTANA, New York J. Med. 58:2525 (Aug. 1) 1958.

Fontana is a pediatrician working in the New York University-Bellevue Medical Center hospitals. Allergens arise from food, inhalation, or infections. All may be present. Intracutaneous tests may give response for foods, pollens, etc., but "are never demonstrated in the purely infective cases.". Cooperation with the rhinologist is desirable. However, the author does not take to "suspicion diagnosis" of adenoids and tonsils as the sole factor in bacterial allergy. He is rather caustic about the surgery done by hit-or-miss operators who are inexpert, and often leave "tags of tissue" or do an incomplete and therefore inadequate operation. Any infected tissue left in situ may activate asthmatic symptoms. It may be good practice to administer antimicrobial therapy before and after operations. A broad spectrum antibiotic (not necessarily penicillin) should be administered. Both specific and nonspecific agents are set forth as helpful but cannot be described here. There are paragraphs dealing with "Education of Parents" and the recognition of emotional factors in asthma of the young.

ABSTRACTER'S COMMENT: Nobody can be quite sure that adenoids and tonsils are infective agents in a given asthmatic. Size of tonsils and amount of adenoids have little to do with the case. Small tonsils may be highly infective. Bacterial cultures do not help as much as one would like in analyzing a situation. There is no reference here to sinusitis in children. Here one may find a potent cause of allergy in children (or adults).

I. W. VOORHEES, New York.

MANDIBULAR MUSCLE SPASMS: THEIR ROLE IN HEAD AND NECK PAIN AND MANDIBULAR DYSFUNCTION. ARTHUR S. FREESE, New York J. Med. 58:2533 (Aug. 1) 1958.

Inability to open or difficulty in opening the mouth is dependent on mandibular immobility. This is often simply functional—seldom organic due to trauma or disease. There is, to be sure, degenerative mandibular joint disease, but it is not common. More often we have to

deal with emotionally disturbed persons. Muscles of mastication are eight: right and left temporal, masseters, and internal and external pterygoids. Symptoms are pain, mandibular dysfunction and emotional disturbance. Such muscle spasms are often associated with grinding the teeth, clenching, and peculiar oral habits. Treatment is essentially psychological. One must get the confidence of the patient and substitute assurance for stubborn inaction. Physical therapy may also help. Prognosis may not be favorable.

I. W. VOORHEES, New York

Books

BOOKS RECEIVED

Ciba Foundation Colloquia on Endocrinology, Volume 12: Hormone Production in Endocrine Tumours (with Cumulative Index to Volumes 1-12). Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Maeve O'Connor, B.A. Price, $9.00. Pp. 351 (incl. index), with 58 illustrations. J. & A. Churchill Ltd., 104 Gloucester Place, Portman Sq., London, W. 1. Little, Brown & Company, Medical Book Dept., 34 Beacon St., Boston 6, 1958.

* **Ein binauraler Hörsynthese-Test zum Nachweis Zerebraler Hörstörungen.** By Priv.-Doz. Dr. Joseph Matzker (with an Introduction by Prof. Dr. H. Leicher). Price, $4.25. Pp. 117, with 46 illustrations. Georg Thieme Verlag, Herdweg 63 (14a), Stuttgart. Intercontinental Medical Book Corporation, New York 16, 1958.

* **Funktionelle Röntgendiagnostik des Mediastinums am Beispiel des Bronchialkarzinoms Demonstriert: Experimentelle Untersuchungen zur Grundlage der Oesophaguskymographie nach Strnad.** By Priv.-Doz. Dr. Med. R. Kraus (with a Foreword by Prof. Dr. F. Strnad). Price, DM 8.40. Pp. 55, with 33 illustrations. Georg Thieme Verlag, Herdweg 63 (14a), Stuttgart. Intercontinental Medical Book Corporation, New York 16, 1958.

Rehabilitation in Industry. (Modern Monographs in Industrial Medicine—Editor in Chief: Anthony J. Lanza, M.D.; Consulting Editor: Richard H. Orr, M.D.) Edited by Donald A. Covalt, M.D. Price, $6.00. Pp. 154, with 31 illustrations. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16. 99 Great Russell St., London, W. C. 1, 1958.

* **Hearing Therapy for Children.** Second, Revised Edition. By Alice Streng, M.A.; Waring J. Fitch, M.A.; LeRoy D. Hedgecock, Ph.D.; James W. Phillips, M.D., and James A. Carrell, Ph.D. Price, $6.75. Pp. 353, with 47 illustrations. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, 99 Great Russell St., London, W. C. 1, 1958.

An Atlas of Esophageal Motility in Health and Disease. By Charles F. Code, M.D., Ph.D.; Brian Creamer, M.D., M.R.C.P. (London); Jerry F. Schlegel, B.S.; Arthur M. Olsen, M.D., M.S. (Med.); F. Edmund Donoghue, M.D., M.S. (Med.), and Howard A. Andersen, M.D., M.S. (Med.) (from the Section of Physiology and the Section of Medicine, Mayo Clinic and Mayo Foundation). Price, $8.50. Pp. 134, with 73 illustrations and charts. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1958.

A Stereoscopic Atlas of Human Anatomy. Section IV: The Thorax (in two volumes: Reels 113-122 and Reels 123-132). By David L. Bassett, M.D. Color Photographs by William B. Gruber. Price, $16.50. Includes 20 Viewmaster reels containing 140 colored stereoscopic views; 140 Black & White line drawings of above, to scale, with labels and keys on 140 facing pages. Sawyer's Inc., Box 490, Portland 7, Ore., 1958.

BOOK REVIEWS

Histopathologie der Tumoren der Kiefer und der Mundhöhle. By Prof. Dr. E. Langer. Price, $11.80. Pp. 167, with 212 illustrations. Georg Thieme Verlag, Herdweg 63 (14a), Stuttgart (American agent—Grune & Stratton, Inc., 381 4th Ave., New York 16), 1958.

This monograph presents a detailed description of the histopathology of the various tumors that may involve the maxilla and the oral cavity. This is purely a pathologic presentation with photomicrographs to clarify the text. The gross appearance of these neoplasms, although described, is not illustrated, nor is there any detailed reference to their clinical implications.

Among the neoplasms of the maxilla, the following are described: adamantinoma, odontoma, enameloma, congenital melanotic tumors, myeloblastoma, osteoma, sarcoma, etc.

Tumors involving the salivary glands include the mixed tumors both benign and malignant, adenoma, cystadenolymphoma, cystadenoma, cylindroma, carcinoma, sarcoma, etc.

* Will be reviewed.

In the oral cavity, the epithelial lesions, tumors of the blood and lymph vessels, of the connective tissue structures, the nerves, the reticuloendothelial system, and the fat and muscular structures are presented.

This monograph is of extreme interest to the pathologist, dentist, oral surgeon, and the rhinologist.

The Esophagus: Medical and Surgical Management. By Edward B. Benedict and George L. Nardi. Foreword by Edward D. Churchill. Price, $15. Pp. 390, with 16 color plates and 108 black and white illustrations. Little, Brown & Company, 34 Beacon St., Boston 6, 1958.

This is indeed a Harvard Book, right out of the Medical School and the Massachusetts General Hospital. The authors are both M.D., F.A.C.S. In his Foreword, Dr. Churchill declares the authors to be "experts" who are here trying to unify under one cover all that has been written about the esophagus in so many different articles, essays, and books on general medicine. He thinks they have succeeded admirably; so do we. They are a bit annoyed at the complacency of the general practitioner who fails to be alerted by the patient's difficulty in swallowing. This may, of course, be the first sign of serious trouble, but not always. In cancer, for instance, there may be no striking symptom until the condition is inoperable. In spite of all the "helps" at hand, early diagnosis of cancer is seldom possible, that is to say, diagnosis which permits early surgery. It is rather surprising to note that perforations are caused by (1) direct tube investigation; (2) bougie; (3) foreign bodies too long "stuck" in the canal. (Would not "lodged" be a nicer word?) It is somewhat distressing to hold the esophagist responsible for perforations. It can mean that by the time he gets his sights on the *locus minoris* it is too late to do much about the lesion present! The esophagologist too often is called in late because there is still a dread among physicians and patients in having "the tube passed." Dr. Benedict as a direct tube man must have this experience often.

Contents of the book are well arranged, beginning with the anatomy and physiology in review, through the various diseases, tumors, obstructions, traumas, hiatus hernias, diverticuloses, varices, malignancies, pellagra, scleroderma, paralyses of peristalsis, and, finally, emotional disorders attending swallowing. It seems that nothing has been left out.

The profuse illustrations in color and black and white are excellent. Frank Netter has made the color plates, working through the Ciba Company, and Mrs. Edith Tagrin has done excellent illustrative work. Without reading much of the text one can get an excellent idea of the contents by "reading the pictures."

Surgery of the esophagus is now well established as to methods and techniques. However, it has its limitations because of complications due to metastases into the mediastinum, extensions which cannot be removed by surgery no matter how aggressive. Transplantation of the colon as a substitute for resection of the esophagus is a brilliant technical achievement, but there are surgeons who prefer to "pull up the stomach" and join its resected end with that of the esophagus which can be allowed to remain. This sounds pretty formidable, but it is being done.

One must mention the very complete references at the end of each chapter. They often run from 20 to 50 in number, and connote an enormous amount of literary research. This should be a great time saver for subsequent authors who have committed themselves to bringing further studies of this kind "up to date." This book is likely to remain a classic in its field for a long time to come.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Eighth International Congress of Bronchoesophagology, Mexico City, March, 1959.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, Miami, March, 1960.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.
Meeting: Atlantic City, June 8-12, 1959.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Place: The Homestead, Hot Springs, Va. Time: March 10-11, 1959 (afternoon only).

American Laryngological Association

President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: The Homestead, Hot Springs, Va. Time: March 8-9, 1959.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: The Homestead, Hot Springs, Va. Time: March 10-12, 1959 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Place: The Savoy Plaza, New York. Time: Jan. 8-9, 1959.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Place: Hotel-Atlanta Biltmore, Atlanta, Ga. Time: Jan. 23-24, 1959.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Place: Park Plaza Hotel, St. Louis. Time: Jan. 12-13, 1959.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.
Place: The Ahwahnee Hotel, Yosemite Valley, Calif. Time: Jan. 17-18, 1959.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.

American Otological Society, Inc.

President: Dr. Moses H. Lurie, 483 Beacon St., Boston.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: The Homestead, Hot Springs, Va. Time: March 13-14, 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.

American Society of Facial Plastic Surgery

President: Dr. Trent W. Smith, 327 E. State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Meetings: March 18 and July 15, 1959, New York.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. E. P. Fowler Jr., 180 Fort Washington Ave., New York 32.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
Secretary-Treasurer: Capt. Maurice Schiff (MC), U. S. N., U. S. Naval Hospital, Oakland, Calif.

Professionally accepted

HALF-INCH SIZE

VASELINE®
STERILE PETROLATUM GAUZE

½" x 72"... in disposable plastic tubes

- ★ Selvage-Edged
- ★ Highly Absorbent
- ★ Lightly Impregnated
- ★ Guaranteed Sterile
- ★ Keeps Indefinitely

Order from your surgical or hospital supply dealer

Sample on request

CHESEBROUGH-POND'S INC.
Professional Products Division
New York 17, N. Y.

VASELINE is a registered trademark of Chesebrough-Pond's Inc.

SPECIALTY INSTRUMENTS

EAR, NOSE, THROAT AND RHINOPLASTIC INSTRUMENTS.

WE SPECIALIZE IN INSTRUMENTS FOR ANY OF THE ABOVE SPECIALTIES.

OUR INSTRUMENTS ARE CORRECT IN PATTERN, HAND FORGED OUT OF SPECIAL STAINLESS STEEL AND EXPERTLY MADE.

HOSPITALS, DOCTORS AND CLINICS HAVE BEEN EQUIPPED WITH OUR SPECIALTY INSTRUMENTS.

BOVIE ELECTROSURGICAL UNITS AVAILABLE.

OUR REPAIR DEPARTMENT CAN SHARPEN, CHROME PLATE OR ADJUST YOUR OLD INSTRUMENTS AND MAKE THEM LIKE NEW.

SEND US YOUR LIST OF REQUIREMENTS AND WE WILL BE GLAD TO QUOTE ON INSTRUMENTS OR EQUIPMENT.

WE CARRY A FULL LINE OF TREATMENT CHAIRS AND EQUIPMENT.

WE EXPORT TO ALL PARTS OF THE WORLD.

QUICK DELIVERIES

Phone, GRamercy 5-0585

S. G. KREBS CO., INC.
351 Second Avenue
New York 10, N. Y.

Distributors for
Bausch & Lomb
Ritter • Reliance
Welch-Allyn
Nat. Elec. Inst. Co.

THE GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL

Announces to the Profession its

THIRTY-SECOND ANNUAL SPRING CONGRESS

in

OPHTHALMOLOGY AND OTOLARYNGOLOGY

APRIL 4 through APRIL 11, 1959

GUEST SPEAKERS

Joseph P. Atkins, M.D.	Philadelphia, Pa.
Walter S. Atkinson, M.D.	Watertown, N. Y.
Kenneth D. Bailey, M.D.	Fairmont, W. Va.
F. Willson Daily, M.D.	Roanoke, Va.
Windson S. Davies, M.D.	Detroit, Mich.
Jeff Davis, M.D.	New York, N. Y.
Francis P. Furgiuele, M.D.	Philadelphia, Pa.
Surgeon Gen. B. W. Hogan, USN	Washington, D. C.
Bayard T. Horton, M.D.	Rochester, Minn.
Wendell L. Hughes, M.D.	Hempstead, N. Y.
John H. King, M.D.	Washington, D. C.
T. Keith Lyle, M.D.	London, England
George T. Nager, M.D.	Baltimore, Md.
Robin M. Rankow, M.D.	New York, N. Y.
Kenneth L. Roper, M.D.	Chicago, Ill.
A. D. Ruedemann, M.D.	Detroit, Mich.
Russell A. Sage, M.D.	Indianapolis, Ind.
Jules G. Waltner, M.D.	New York, N. Y.
Edgar N. Weaver, M.D.	Roanoke, Va.
Walter L. Winkenwerder, M.D.	Baltimore, Md.

For Further Information Write:

Superintendent, P. O. Box 1789, Roanoke, Virginia

Of interest to you and your patients

THE MENACE OF ALLERGIES

WHAT WE KNOW ABOUT ALLERGY
by Louis Tuft, 12 pages, 15 cents

HOUSE DUST ALLERGY
by Karl D. Figley, 8 pages, 15 cents

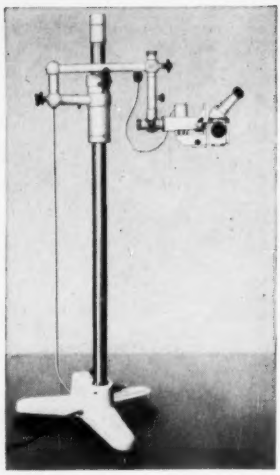
FOOD ALLERGY
by Samuel M. Feinberg, M.D., 6 pages, 10 cents

SKIN ALLERGY
by Samuel M. Feinberg, M.D., 6 pages, 10 cents

ASTHMA AND HAY FEVER
by Samuel M. Feinberg, M.D., 6 pages, 10 cents

RAGWEED AND HAY FEVER
by Oren C. Durham, 2 pages, 5 cents

AMERICAN MEDICAL ASSOCIATION
535 NORTH DEARBORN STREET • CHICAGO 10 • ILLINOIS



STAPES MOBILIZATION

MYRINGOPLASTY—TYMPANOPLASTY

The Instruments of:

ROSEN • SHAMBAUGH & DERLACKI • KOS • HOUSE • GUILFORD & WRIGHT • WULLSTEIN • ZOELLNER • HEERMANN and others

plus the ZEISS OTOSCOPE MICROSCOPE

With all accessories, including sterilizable stainless shields for eye pieces, objectives. Photographic attachments available, too.

Write For Illustrated Booklet . . .
Free on Request

V MUELLER & CO.
330 South Honore Street
Chicago 12, Illinois

DALLAS • HOUSTON • LOS ANGELES • ROCHESTER, MINN.

RELIEVE PAIN

SINUSITIS

REDUCE SEVERITY

"Acts by Suction"

is an infection which rarely remains localized and if untreated often causes serious tissue destruction. Nasopharyngeal antisepsis as accomplished with the

Nichols NASAL SYPHON

after FORTY-ONE years has become routine practice in many clinics oftentimes obviating the necessity of surgical intervention. It quickly allays SINUS PAIN due to congestion as it removes the cause—inspissated mucopus in the sinus openings —thus re-establishing necessary DRAINAGE and VENTILATION. Send in coupon for detailed information.

NICHOLS NASAL SYPHON, INC. MONTCLAIR, N. J.
Please send details of SPECIAL OFFER "S" without obligation.

NAME..... M.D.
ADDRESS.....
CITY..... ZONE..... STATE.....

9 SPECIALTY JOURNALS

PUBLISHED MONTHLY

BY THE AMERICAN MEDICAL ASSOCIATION

NEUROLOGY AND PSYCHIATRY

DISEASES OF CHILDREN

INTERNAL MEDICINE

INDUSTRIAL HEALTH

OTOLARYNGOLOGY

OPHTHALMOLOGY

DERMATOLOGY

PATHOLOGY

SURGERY

each journal offers
the latest medical findings by
outstanding authorities in
its special field . . .
of value not only
to the specialist but
to the general practitioner as well

to order your subscription to one of the A.M.A.'s
specialty journals use the form below

AMERICAN MEDICAL ASSOCIATION
535 North Dearborn • Chicago 10

Please enter my subscription to the specialty journal checked at right.
Remittance for ☐ one year ☐ two years is enclosed.
Start my subscription with the next Issue.

NAME_____

ADDRESS_____

CITY_____ ZONE____ STATE_____

	U.S.A. & Possessions APO's	Canada	Outside U.S.A. & Possessions
☐ A.M.A. Arch. Neurology and Psychiatry	$14.00	$14.50	$15.50
☐ A.M.A. Arch. Dermatology	12.00	12.50	13.50
☐ A.M.A. Arch. Industrial Health	10.00	10.50	11.50
☐ A.M.A. Arch. Internal Medicine	10.00	10.50	11.50
☐ A.M.A. Jrl. Diseases of Children	12.00	12.50	13.50
☐ A.M.A. Arch. Surgery	14.00	14.50	15.50
☐ A.M.A. Arch. Pathology	10.00	10.50	11.50
☐ A.M.A. Arch. Ophthalmology	12.00	12.50	13.50
☐ A.M.A. Arch. Otolaryngology	14.00	14.50	15.50

Chair—Upholstered, reclining ..$150.00

Cabinet—Stainless steel, eight drawers with or without complete transilluminator, cautery rheostat and transformer, waste container, air regulator, gauge, tubing and cutoff ..$115.00 to $195.00

Light—Telescopic .....$ 16.50

Cuspidor—With Suction .....$ 95.00

Stool—Upholstered, with easy running ball-bearing casters ..$ 20.00

Light Shield .....$ 5.00

All equipment shown in the above reproduction (with spray bottles, stainless steel cotton jars, ready to be attached to your compressed air system, $531.50 F.O.B. Factory.

Catalogue sent on request

1901-1905 Beverly Blvd. SMR Los Angeles 57, Calif.

"Doctor, will you tell me..."

What are the symptoms?
How shall I treat it?
What causes it?

For practical information about communicable disease, have your patients read:

SCARLET FEVER
8 pages, 15 cents

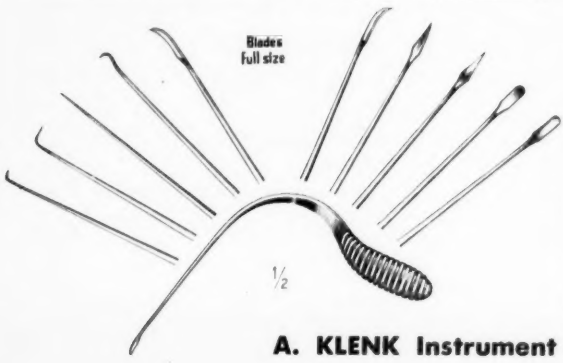
IMMUNIZATION
by Ruth A. Thomas, 8 pages, 15 cents

WHOOPING COUGH
by Constance Frick, 4 pages, 10 cents

PNEUMONIA'S WATERLOO?
by William W. Bolton, 12 pages, 15 cents

WE CAN PREVENT DIPHTHERIA
by P. S. Rhoads, 8 pages, 15 cents

AMERICAN MEDICAL ASSOCIATION • 535 NORTH DEARBORN • CHICAGO 10



Zoellner's
SET OF lo TYMPANOPLASTY INSTRUMENTS $70.00

from left to right:
fine hook, short, curved upward
fine hook, long, curved upward
straight needle, fine
fine hook, curved, downward
fine knife, downward cutting
fine knife, upward cutting
fine needle curved to the *left*
fine needle curved to the *right*
fine raspatory curved to the *left*
fine raspatory curved to the *right*

LATEST TYPE Rosen set for stapes mobilization including new curettes, needles & perforator $45.0♥

A. KLENK Instrument Co. 60-17 70 St. Maspeth 78, L. I., N. Y.

CAMERON SURGICAL SPECIALTY COMPANY

SALES AGENCIES IN LEADING CITIES THROUGHOUT THE WORLD

CABLE ADDRESS
"CAMSURGCO" - CHICAGO

NEW YORK AGENCY
FISK BUILDING
57TH & BROADWAY

CAMERON'S ELECTRO-DIAGNOSTIC, SURGICAL
AND DENTAL INSTRUMENTS — CAUTERY
EQUIPMENT AND OPTICAL SPECIALTIES

666 W. DIVISION STREET
CHICAGO 10, U. S. A.
MIchigan 2-7425

Dear Doctor:

This is a report from your Meeting.

Prior to the Chicago meeting, you received a folder describing the new Cameron electro-surgical unit and accessories designed for your specialty. We also described our new binocular loupes.

Many who did not attend the meeting ordered these units on approval and confirmed their satisfaction with a check.

At the convention, YOUR ORDERS DOUBLED last year's sales. We are proud that the new Cameron units fill a need in your practice, as proved by the avalanche of orders. We appreciate the time you spent at our booth examining the new diagnostic and electro-surgical equipment.

Thank you.

If you did not stop at our exhibit or haven't seen our New Model 255 circular, please drop me a note. Or better still, request a unit on approval and see how useful it is in your practice. With the EENT model of the new Cameron No. 255 electro-surgical unit, you can perform over 72 operations in your office. Yet the price range is only $196.50 to $288.50 complete, depending on accessories.

Thank you again for your enthusiastic response. Write today for the new 255 circular.

Sincerely,

Noel Wallen

Noel Wallen
President

DeVilbiss ANNOUNCES

Steam • dial

STEAMS 'ROUND THE CLOCK

NOW FOR THE FIRST TIME YOUR PATIENT CAN DIAL THE EXACT AMOUNT OF STEAM YOU PRESCRIBE

NO. 142

DeVilbiss Steam • dial

RETAIL **$9.95**

Complete with cord and stand

Other DeVilbiss Vaporizers from $3.95

Here is an entirely new concept in vaporizers, engineered and produced by DeVilbiss to take the guesswork out of steam therapy.

With the simple twist of a dial, the proper steam volume may be selected for any size room and for the specific needs of the patient, whether infant, child or adult.

Hard water poses no problems for STEAM • DIAL – no more spitting or sputtering. Just plug in the cord, and within minutes you can set the dial for the precise steam output needed.

Prescribe the DeVilbiss STEAM • DIAL, the only vaporizer-humidifier on the market with a volume control dial.

SAFE — Shuts off automatically when water reaches safety level.

STEAMS ALL NIGHT — Water capacity for 'round the clock operation at maximum setting.

GUARANTEED BY DEVILBISS

For gently medicated steam recommend DeVilbiss Benz-O-Gel, the non-gumming benzoin base compound for use with steam Vaporizers.

SINCE 1888, THREE GENERATIONS OF PHYSICIANS HAVE USED AND PRESCRIBED DEVILBISS

DeVilbiss • Somerset, Pa.

freedom . . .

no more restraining older children in infant type humidity units . . . At last, with the new Walton Model HA the patient—child or adult—has complete freedom of the bed while undergoing Cold Steam* humidity treatment; the Model HA humidity canopy encompasses the entire bed—makes each bed a humidity room in itself. The patient can be treated with correct amount of moisture required, for the flow of moisture can be regulated. The same unit may also be used as a room humidifier and to increase humidity in oxygen tents. Sold through leading hospital supply dealers, or write

*Trademark.

Walton

LABORATORIES, INC., IRVINGTON 11, N.J.

Chicago Office: 548 W. Washington Boulevard